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INTRAVENOUS INFUSIONS IN INFANTS—THE GARDNER-MURPHY TECHNIQUE

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and

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A recent note[1] on a method of using polythene for intravenous infusions in adults, has prompted us to submit this account of our method of utilizing polythene tubing in order to simplify the technical side of intravenous infusions in infants.

The use of the Gardner-Murphy combination needle[2] (G-M needle) was first reported in 1950, and in 1953 one of us (S.L.) learnt the technique from Abella[3] who noted its value in that year. It has been used on many hundreds of children over the past 3 years. In the children's wards of this hospital the G-M needle has been in use since July 1956; from that time until the end of December 1956, well over 100 intravenous infusions have been put up on babies (mostly suffering from gastro-enteritis), and of these, only 5 or 6 had to be done with a cut-down, most of the rest being usually easily set up by means of the G-M needle.

Briefly, the G-M combination consists of a proximal size-24 BD needle inserted into a piece of size-1 polythene tube of about 10 inches in length. For the distal end of the tube, the shaft of a size-24 BD needle is used. The shaft of a BD needle simply broken off at the hub comes away quite freely and remains patent. About a quarter of the shaft is inserted snugly into the distal end of the polythene. The needle shaft, held by an artery forceps (Fig. 1), is then ready to be inserted into an accessible vein (Fig. 2) and taped down with a few strips of strapping previously made ready for this purpose. We prefer to use scalp veins and when these are used, there is no need to splint the head, but if the baby is restless it is advisable to restrain the arms, or the drip may be pulled out.

The back of the hand is also a useful place for a 'push-in', as is also the volar aspect of the wrist. Generally speaking it is not advisable to fix an extremity in a splint before inserting the needle in a vein. It is better first to set up the

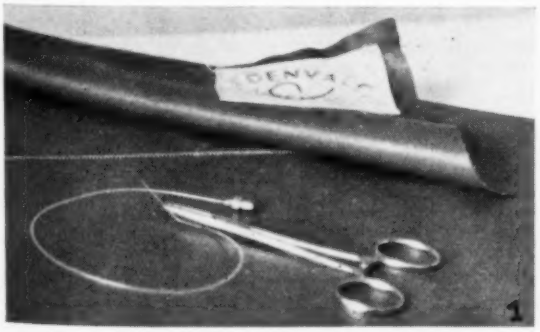

Fig. 1. The G-M unit. Distal needle held by forceps.

Fig. 2. The vein about to be entered. Note proximal G-M needle separate from drip tubing.

drip, and then to splint the extremity. Conventional splints are often too cumbersome. We prefer to use a wooden spatula, which works admirably. When the needle has been taped in place and the infusion is running, *two* spatulae (padded with cotton wool) are used as splints. They are placed one on either side of the forearm and taped together with zinc-oxide strapping.

The actual insertion of a needle into a vein requires no great skill. The experienced operator will, however, often find veins where the less experienced would not think that any such existed. Thus, for example, probably the best veins of all for an intravenous needle are the two small veins, commonly invisible, which run down vertically from the region of the anterior fontanelle towards the nasion (Fig. 2). These veins can often be made to appear by obstructing their flow at the nasal end and gently flicking or pressing over the vein. A further advantage is that when these veins are used the scalp need not be shaved.

The real difficulty is retaining a needle in place. In the conventional method the hub of the needle and the dead weight of the adjacent drip apparatus are factors which tend to dislodge a needle, especially if there is much movement of the head. These are difficult to strap down onto the scalp to keep them secure. With the use of the G-M combination, retaining the needle in place is no problem whatever. The needle is taped down with two small pieces of strapping (Fig. 3) and will give no trouble at all.

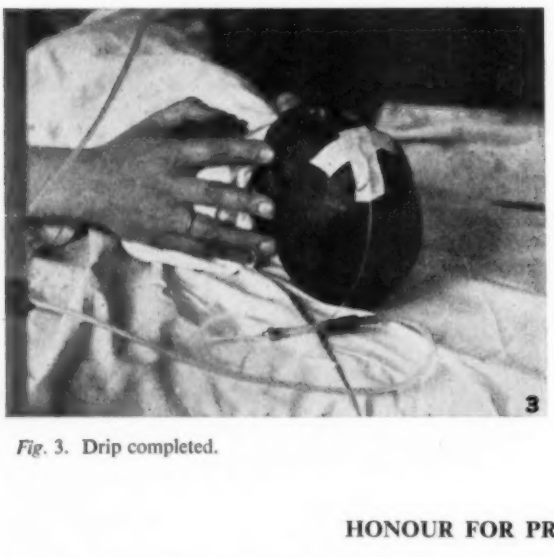

Fig. 3. Drip completed.

Sterilization. The needle combination is best sterilized by keeping it in a 1 : 1,000 solution of Zephiran. It should be rinsed out with the intravenous fluid in order to flush out the Zephiran before use. In so doing one also notes whether or not there are any leaks at the junction of needle and polythene. The patency of the needles is also ensured, and a distal needle partially blocked because of late or inefficient flushing out after previous intravenous use can be rejected. This obstruction interferes with the blood reflux when a vein has been entered, and if there is a suspicion of partial blockage a new needle shaft should be inserted.

The proximal end of the G-M unit should hang free, and be disconnected from the drip tubing whilst the distal needle, held be forceps, is inserted into the vein. In this way, when the vein is entered, reflux of blood into the polythene tubing is immediate. The drip is then connected and allowed to run.

The internal calibre of No. 1 polythene often varies from batch to batch, and it is important to select tubing which will fit tightly onto the size-24 needle. Should the fit not be tight, the ends of the polythene tubing must be stretched so as to diminish the internal calibre before the needle is mounted.

Size-24 BD needles are the smallest size through which blood and plasma will flow with little difficulty. At the conventional drip-stand height, clear fluid will flow through at a rate up to 20 drops (2 c.c.) per minute.

Ideally, the point of the proximal needle should be cut off, for it tends to pierce the polythene tubing. Alternatively, this can be circumvented by using an extra long piece of polythene, so that the proximal needle can be strapped down to the bed and prevented from moving.

We thank Dr. J. D. Prestwich, Superintendent of Edenvale Hospital, and Dr. B. Epstein, paediatrician, who kindly gave permission to publish this report.

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HONOUR FOR PROFESSOR DRENNAN

The South African Association for the Advancement of Science has announced that the South Africa Medal and Grant for 1956-1957 has been awarded to Prof. Matthew Robertson Drennan, who for 36 years occupied the Chair of Anatomy at the University of Cape Town till his retirement as Professor Emeritus in 1955.

In making the award the Association states: 'During 43 years of almost continuous service in the South African College and Cape Town University no single personality has impressed his stamp more profoundly upon the first Medical School in South Africa, and upon the more than 2,000 Medical practitioners that have issued therefrom than has Matthew Robertson Drennan.'

Professor Drennan has written many medical books and has been responsible for a number of medical discoveries in several fields following his researches among African people and animals.

Directly influenced and inspired by his teaching are several medical authorities in different parts of the world.

OBITUARY

HENRY MEREDITH GRIFFITHS, F.R.C.S. (EDIN.)

We regret to announce the death of Dr. Henry Meredith Griffiths, F.R.C.S. (Edin.), late Medical Officer at the Dynamite Factory Somerset West, which occurred on 31 March.

An In Memorial Notice will appear in a later issue of the *Journal*.

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VAN DIE REDAKSIE

PROGNOSE BY KANKER

Crile[1] het onlangs, uit 'n oorwegend kliniese oogpunt, die faktore, wat by die verspreiding van kanker betrokke is, ontleed, met spesiale verwysing na kanker van die bors.

Die verspreiding van 'n karsinoom word grootliks bepaal deur die biologiese tipe of klas waaraan dit behoort en deur die weerstand van die pasiënt. Die oorspronklike opvatting dat alle kankers as 'n gelokaliseerde siekte begin en mettertyd versprei om die streeklimfkliere en nog later die bloedsomloop in te sluit, is nie heeltemal juis nie. Soos Crile aandui, is 'n basaalselkarsinoom van die vel 'n biologiese tipe wat geneig is om slegs lokaal in te dring. 'n Skaalvormige epitelioom van die vel matastaseer tot die streeklimfknope en baie selde sistemies. Melanome mag sistemies versprei selfs voordat die primêr opvallend is. Hierdie is konstante biologiese tipes en wanneer hulle as sulks erken is, kan hulle daaropvolgende ontwikkeling en wyse van verspreiding, om Crile se analogie te gebruik, op dieselfde wyse voorspel word as ons die onbekwaamheid van 'n skilpad om in 'n haas te verander, herken. Die prognose sal dus afhang van die kwaadaardige vermoë van die besondere biologiese tipe waarmee ons handel en ongelukkig is histologiese ondersoek, ten spyte van uitvoerige graderingsmetodes, nie altyd 'n akkurate manier om 'n gewas aan sy juiste biologiese klas toe te wys nie. Op dieselfde wyse waarop verskillende kankers van die vel verskillende verspreidingsvermoëns aan die dag lê, is dit redelik om te veronderstel dat kankers van die bors en inwendige organe hulle op dieselfde wyse sal gedra. Hierdie begrip, tesame met 'n bewustheid van die beperking van histologiese ondersoeke, mag die teenstrydighede en weersprekings verklaar wat so dikwels aangetref word wanneer artikels oor borskanker noukeurig bestudeer word.[2]

Lank reeds is die leer verkondig dat hoe gouer die kanker gediagnoseer word, hoe groter die kans is om 'n kuur te bewerkstellig. Crile, McKinnon en andere het onlangs twyfel gewerp oor die waarheid hiervan. McKinnon[3] het getoon dat, ten spyte van kankerbeheermaatreëls in sekere provinsies van Kanada, waar intensiewe veldtogte onderneem is om die publiek in vroeë diagnose te onderrig, was daar geen afname in die sterftesyfer weens kanker wat volgens ouderdomsgroepe bepaal is nie. Hy het ook gevind dat die sterftesyfer in sommige Kanadese provinsies, in Massachusetts en in Engeland en Wallis, feitlik dieselfde is ten spyte van verskille in maatreëls vir kankerontdekking. Crile sê heel pertinent dat ‚as vroeë diagnose en radikale behandeling 'n gegronde antwoord tot die probleem van kanker is, kan 'n afname in die sterftesyfer verwag word op dieselfde wyse waarop vroeë diagnose en behandeling die sterftesyfer weens tuberkulose verminder het'. Bloom,[4]

EDITORIAL

PROGNOSIS IN CANCER

Crile,[1] has recently analysed, from an essentially clinical point of view, the factors concerned in the spread of cancer, with special reference to cancer of the breast.

The dissemination of a carcinoma is largely determined by the biological type or class to which it belongs, and the resistance of the host. The original concept that all cancers start as a localized disease, and then, with the passage of time, spread to involve the regional nodes and still later the blood stream, is not entirely accurate. As Crile points out, a basal-cell carcinoma of the skin is a biological type which tends to remain only locally invasive. A squamous epithelioma of the skin metastasizes to the regional nodes and rarely systemically. Melanomas may spread systemically even before the primary is noticeable. These are constant biological types and when recognized as such, their subsequent development and mode of dissemination can, to use Crile's analogy, be predicted in the same way as we recognize the inability of a tortoise to change into a hare. The prognosis, then, will depend on the malignant potential of the particular biological type with which we are dealing and, unfortunately, histological examination, despite elaborate methods of grading, is not always an accurate means of assigning a tumour to its correct biological class. In the same way as different cancers of the skin manifest different potentialities for dissemination, so may it be reasonable to suppose that cancers of the breast and internal organs behave in a similar fashion. This concept, together with an awareness of the limitations of histological examination, might explain the inconsistencies and contradictions so often found in perusal of breast-cancer literature.[2]

It has long been the teaching that the earlier cancer is diagnosed, the greater is the chance of effecting a cure. Crile, McKinnon and others have recently cast doubt on the truth of this. McKinnon[3] has shown that despite cancer control programmes in certain provinces of Canada, where intensive campaigns are undertaken to educate the public in early diagnosis, there has been no decrease in the age-adjusted death-rate from cancer. He has also found that the mortality rate in some Canadian provinces, in Massachusetts, and in England and Wales, is practically identical despite differences in cancer detection programmes. Crile pertinently remarks that if 'early diagnosis and radical

na 'n studie van borskanker in Engeland, het verklaar dat die oorlewingsyfer dieselfde is, afgesien van die lengte van die geskiedenis. Park en Lees[5] het aan die hand gedoen dat behandeling inderdaad nie die oorlewingsyfer verander nie en hierdie mening is deur Crile en andere aangehaal om die betreklike magteloosheid van chirurgiese ingryping by borskanker aan te dui. Soos Hedley Atkins[6] aangedui het, het hierdie skrywers tot hulle gevolgtrekking gekom na 'n statistiese oorsig van die resultate van ander navorsers sonder dat hulle enige klaarblyklike praktiese kontak met die siekte gehad het. Kraus,[7] deur gebruik te maak van Park en Lees se eie syfers, het tot presies die teenoorgestelde gevolgtrekking gekom, nl. dat behandeling inderdaad die oorlewingsyfer wysig. Atkins egter, by bespreking van die relatiewe voordele van radikale borsverwydering in teenstelling met McWhirter se stelsel van eenvoudige borsverwydering, en radioterapie toegespits op die ongedissekteerde oksel, verklaar , ... The difference in survival rates between treatment and no treatment is so small that the difference between one method and another must inevitably be smaller still. Different treatment may make little difference, but how much that little difference means to the little group who differ!'

Behalwe die moontlikheid van verspreiding wat by die gewas self aanwesig is, het dit onlangs duidelik geword dat die chirurg onder sekere toestande self verantwoordelik mag wees vir die verspreiding van die gewas. Fisher en Turnbull[8] het getoon dat manupilasie van 'n rektale karsinoom by operasie mag veroorsaak dat 'n storting van selle die bloedsomloop binnegaan, en Goligher *et al.*[9] het gedemonstreer dat inplanting mag geskied deur selle wat los in die binneruimte van die ingewande lê ten tye van 'n kolonreseksie. Deur middel van smere het Crile gedemonstreer dat daar by 10% opereerbare kankers van die kolon kwaadaardige selle op die buikvliesoppervlakte aanwesig is en dat selfs 'n ondersoekingslaparotomie verspreiding deur hantering alleen, mag veroorsaak. Om hierdie rede is hy nie heeltemal ten gunste van die ‚second look'-operasie van Wangensteen nie.

Dit wil voorkom, ten spyte van die uitgesproke menings van sekere skrywers dat daar geen belangrike verbetering in die algemene oorlewingsyfer van kanker as gevolg van vroeë diagnose en behandeling was nie, dit nie verstandig is om die pleidooi om vroeë diagnose te laat vaar nie aangesien dit bewys is dat vroeë opsporing by sekere tipes van kanker van nut is. Om Crile te siteer: ‚It would be astonishing if there were no truth in the conventional concept of the value of early and radical excision of cancer, and no thoughtful surgeon can question the value of early eradication of pre-invasive cancer. The challenge to surgeons is to preserve the good of prompt and radical surgery, to use it when effective, and to find a way of avoiding operations that are useless and do nothing but harm'.

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treatment were a valid answer to the problem of cancer, a reduction in the death-rate might have been expected in the same way that early diagnosis and treatment has reduced the death-rate from tuberculosis'. Bloom[4] from a study of breast cancer in England has stated that the survival rate is uniform, no matter how long the history. Park and Lees[5] have suggested that treatment does not in fact alter survival rate at all, and this opinion has been quoted by Crile and others to indicate the relative impotence of the surgical attack on cancer of the breast. As Hedley Atkins[6] has pointed out, these writers arrive at their conclusion by a statistical survey of the results of others without any apparent practical contact with the disease at all. Kraus,[7] using Park and Lees' own figures, arrived at an exactly opposite conclusion, viz. that treatment does in fact alter the survival rate. Atkins, however, in discussing the relative merits of radical mastectomy as opposed to McWhirter's regime of simple mastectomy and radiotherapy directed to the undissected axilla, states ' . . . . The difference in survival rates between treatment and no treatment is so small that the difference between one method and another must inevitably be smaller still. Different treatment may make little difference, but how much that little difference means to the little group who differ!'

In addition to the potentiality for dissemination possessed by the tumour itself, it has recently become apparent that under certain circumstances the surgeon himself may be responsible for spreading the tumour. Fisher and Turnbull[8] have shown that the manipulation of a rectal carcinoma at operation may cause a shower of cells to enter the bloodstream, and Goligher *et al.*[9] have demonstrated that implantation may occur from cells lying free in the lumen of the bowel at the time of colon resection. Crile has demonstrated by means of smears that 10% of operable colon cancers have malignant cells present on the peritoneal surface, and that even an exploratory laparotomy may cause dissemination from handling alone. For this reason he is not entirely in favour of the 'second look' operation of Wangensteen.

It would appear, despite the expressed views of certain writers, that there has been no significant improvement in the general survival rates of carcinoma as a result of earlier diagnosis and treatment, that it is not reasonable to abandon the plea for early diagnosis, since early detection has been of proved benefit in certain types of cancer. To quote Crile: 'It would be astonishing if there were no truth in the conventional concept of the value of early and radical excision of cancer, and no thoughtful surgeon can question the value of early eradication of pre-invasive cancer. The challenge to surgeons is to preserve the good of prompt and radical surgery, to use it when effective, and to find a way of avoiding operations that are useless and do nothing but harm'.

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CUSHING'S SYNDROME—A PITUITARY OR AN ADRENAL DISEASE?

Bilateral hyperplasia of the adrenal glands is the commonest proximate cause of Cushing's syndrome. Hydrocortisone is secreted in excess, and this glucocorticoid over-production may account for the whole galaxy of symptoms associated with the syndrome. When the disease was first described it was believed to be caused by basophil adenoma of the pituitary gland. We now know, however, that not all cases of Cushing's syndrome are associated with such a tumour, while the characteristic hyaline change in the basophil cells can be produced by exogenous hydrocortisone, and therefore may represent the result of Cushing's syndrome rather than its cause. Nevertheless, a bilateral hyperplasia, unlike an autonomous tumour, suggests the presence of some basic stimulating agent, which brings us back to the corticotrophin of the pituitary gland. (By analogy, however, it may be mentioned that hyperplasia of all four parathyroid glands can occur without any known stimulating agency.)

Other arguments in favour of a pituitary origin for the syndrome include the fact that a fair proportion of patients undergo at least a partial remission as the result of treatment directed at this gland, whether irradiation, electro-coagulation, or surgical ablation. With the first two methods of treatment this remission is produced without any clinical or chemical evidence of hypopituitarism being found. A further point is the frequent finding that cortisone (and now the newer steroid products such as prednisone and fluorohydrocortisone) will actually reduce the output of adrenal steroids in Cushing's syndrome with bilateral hyperplasia. These substances are known to act on the pituitary and to inhibit ACTH production in other circumstances.

Arguments against the theory of the primary over-production of pituitary ACTH are even more cogent. No excess of this hormone can be found in Cushing's syndrome, whereas it has constantly been reported to be present in Addison's disease, in which hydrocortisone, the natural inhibitor of ACTH production, is grossly reduced. Moreover, an injection of ACTH in a patient with Cushing's syndrome frequently produces a further large outpouring of adrenal steroids, which would not be expected if the adrenals were already maximally stimulated by endogenous corticotrophin. Finally, chronic administration of corticotrophin experimentally fails to induce the hyper-response of the adrenal cortex which is fairly constantly found in the hyperplasia type of Cushing's syndrome.

Jailer *et al.*[1] mention several other, more intricate, observations of their own, some supporting a pituitary and some an adrenal origin for the syndrome. They go on to suggest a way round this impasse. They imagine the primary fault to be in the pituitary gland, not in the over-production of ACTH, but in the production of an abnormal potentiator of ACTH. Such a hypothesis certainly seems to fit the facts well and in particular may explain why an excess of circulating corticotrophin, *as estimated by present methods*, is not found.

There is evidence that such 'corticotrophic factor' does exist, distinct from the ascorbic-acid-depleting factor or steroidogenic corticotrophin. This factor, experimentally, has been shown to potentiate the steroidogenic effect of corticotrophin in human subjects. The big difficulty is that such a potentiating substance has not yet been found in Cushing's syndrome, nor is there any more direct evidence for it. The theory is as yet no more than an endocrinologist's arm-chair speculation, but it is worth considering for the future.

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TUBERCULOSIS AS A POSSIBLE AETIOLOGICAL FACTOR IN MENTAL DISORDER, AND THE PLACE OF ISONIAZID IN TREATMENT

WITH FIVE ILLUSTRATIVE CASE REPORTS

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In working for several years in the non-European male wards of a large mental hospital, I encountered a considerable number of patients suffering from tuberculosis both amongst the new admissions and those who had been detained for some time. In these cases we relied on the opinion of a hospital for chest diseases in the assessment of the tuberculous condition and in treatment; account was taken of regular temperature and weight charts, erythrocyte sedimentation rate, chest X-rays, and physical examination. In each case the psychiatric condition was assessed in the normal routine manner.

During the months, it was observed that one case after another improved psychiatrically as he improved physically, and this while he was receiving treatment for the physical disease only. Moreover, these cases were found to present similar psychiatrical syndromes, all conforming to the picture of a toxic confusional syndrome with hallucinatory experiences and irrational, restless and often maniacal behaviour.

These cases should be differentiated from the majority of our cases suffering from tuberculosis, viz. those with established mental diseases, e.g. schizophrenia, dementia paralytica, senility, etc., who have also at some stage or other contracted tuberculosis. In these people also there may be important interrelationships between the diseases, but with this we are not at present concerned.

The first-named, smaller group, however, did seem to illustrate that tuberculous toxaemia *per se* is capable of causing mental disorder. In reviewing the literature, one was surprised that descriptions of this direct relationship

were hard to find. In this paper, therefore, a short review of the literature is made, and 5 cases illustrating this aetiological factor in mental disease are presented.

A further interesting facet to this concept is also dealt with, viz. that isoniazid, one of the most potent drugs available for combating tuberculosis, has been regarded as having strong toxic effects on the nervous system, and *inter alia* causing mental disorder. The advisability of using it in the face of existing disorder is, therefore, often questioned. My own experience, however, has lent no support to this view. The literature of this aspect is reviewed, and it is shown that some of our cases improved dramatically—and stayed well—both mentally and physically while they were subjected to isoniazid administration.

REVIEW OF LITERATURE

Well-nigh every text-book on psychiatry mentions organic disease as a possible aetiological factor in mental disorder. Toxic, infective and degenerative conditions are usually listed amongst others, and the resultant psychiatric syndrome is labelled 'the organic reaction type'. This is characterized clinically by confusion and disorientation, with varying degrees of impairment of intellect, memory and personality. The condition may run an acute or more chronic course, the patient may show gross emotional lability, and at times irrational, uncontrolled behaviour—often in answer to distressing hallucinatory experiences, which may be auditory, visual or haptic in type.

On the more specific relationship between tuberculosis and insanity, a great deal has been written—as ably reviewed by Merrill[1] in 1953. Numerous attempts were made to establish a common aetiological basis, and it was even stated that the 'shut-in' personality types developed schizophrenia on a psychological level, and tuberculosis on a physiological level.[2] Similarly, some workers hunted for a common physiological basis, and investigations were for instance directed at the autonomic nervous system,[3,4] while others tried to establish a common hereditary basis.[5]

Another aspect that has received attention in the literature is that of psychological complications of chronic tuberculosis, especially in patients hospitalized for long periods.[6,7] Again, there were attempts at formulating personality types common to all tuberculotics, and they were variously described as being basically elated ('spes phthisica'), depressed, regressed, hostile, undisciplined, and even over-sexed.

In one psychological study[8] of 100 patients in a tuberculosis sanatorium, however, the authors conclude that 69% were normal, 7% showed a simple maladjustment, 20% were neurotic and 4% psychotic. They say, moreover that, 'no abnormal state specific to tuberculosis has been discovered'. This does, of course, not mean that a toxic confusional form of insanity may not follow tuberculosis; such cases probably usually present with the more striking features of the psychosis, and are therefore to be looked for in mental hospitals rather than in sanatoria.

Another author[9] was concerned with the direct causative interrelationship between the two conditions, and published a small series of 5 cases in this respect. He states in his summary: 'Five cases of mental disturbance associated with pulmonary tuberculosis are described, and an attempt made to elucidate the connection between these two conditions. In the first 3 it is concluded that a mental instability was present previously and that the tuberculosis was merely a means of bringing it to light and was in no way the direct cause; while in the remaining 2 cases the tuberculous toxaemia and their own special circumstances combined to produce an acute confusional insanity which cleared up with an improvement in their physical condition'.

The series of five cases to be described in this article, finds a direct link with the last 2 cases described above.

Recently medical science has been enriched by the knowledge that the hydrazine derivatives of isonicotinic acid are strongly effective in the treatment of active tuberculosis. The rather muddled concept of the relationship between tuberculosis and insanity, was now further complicated by the reports that these new drugs had specific toxic effects on the nervous system. These reports were made in 1952, when detailed preliminary work on the pharmacology and toxicology of the new drugs was being conducted. Thus it was established[10] that the acute LD_{50} dose (for dogs) of isonicotinic acid hydrazide (isoniazid, INH) by intravenous injection was 50 mg. per kg. of body-weight, and that death was due to convulsions. Similarly, the maximally tolerated chronic dose was 10 mg./kg. per day. Again, it was established[11] that the Rhesus monkey developed convulsions after a single dose of 80 mg./kg., and tolerated doses of 20 mg./kg./day for over 7 months.

Apart from the convulsions (leading to death) these articles reported anorexia, ataxia, and excitement as side-effects of INH. Soon afterwards, reports were published on the effects of the drug on humans. Thus [12,14] increased deep reflexes, drowsiness, tremor, twitchings, constipation, restlessness, irritability, excessive energy, elation, nausea and generalized 'nervousness' were reported, as well as convulsions, especially in patients with previous histories of epilepsy. It is evident that toxic effects on the nervous system caused the most concern.

Of all these numerous toxic signs, that of neuritis has been most consistently described. This often presented first with hyperreflexia and paraesthesiae, and later developed the full picture of peripheral neuritis.[13-18] Just as consistently, however, these workers reported that the neuritis was reversible on cessation of the drug. It was specifically mentioned by some that the incidence of neuritis increased with higher doses of INH. It was found for instance to occur in 44% of patients receiving 16-24 mg./kg./day for at least 4 weeks.[16]

In this same article the authors refer to a possible relationship between INH and pyridoxine metabolism and report that pyridoxine administration diminishes the hazard of neuritis, whilst it does not interfere with the therapeutic effectiveness of the drug. This relationship between INH toxicity and vitamin deficiency has received widespread attention. Thus[19] a case was reported in which the patient developed overt signs of pellagra as regards skin, mucous membrane and mental condition while receiving 8 mg./kg. of INH per day for less than 3 weeks. He had been normal in all these respects before isoniazid administration and improved dramatically on cessation of this drug and the administration of 4-hourly doses of nicotinic acid, 40 mg. intramuscularly and 50 mg. orally, and vitamin-B complex intramuscularly twice daily.

A similar case was described by Zabad.[20] The same picture of mental disturbance together with hyper-reflexia developed, and again it was reversible on cessation of INH

and the prompt administration of nicotinamide (50 mg. orally every 6 hours). Here however, there were no signs of pellagra in the skin or mucous membrane. Zabad concludes that the administration of nicotinamide might be indicated as a prophylactic measure against the toxic effects of INH on cerebral functions.

The latest contribution in this respect was made recently by Harrison,[21] who describes a case developing typical pellagrous dermatitis while receiving 300 mg. of isoniazid daily. This woman's food-intake had been poor for several years before, but she only developed pellagra after the administration of isoniazid, and although she was receiving pyridoxine prophylactically. It is suggested that isoniazid acts as an antagonist of nicotinamide (which it closely resembles structurally), and that a pre-pellagra patient—as this one was—is especially liable to develop pellagra while being given isoniazid. This contention is borne out by the fact that the condition remained unchanged for 3 weeks after cessation of isoniazid, but was dramatically reversible when nicotinamide was administered. The exact mechanism of the antagonistic action (for example, whether it occurs because of interference with essential co-enzyme systems) has not yet been established clearly. It does, however, seem fair to conclude that signs of toxicity to the peripheral and central nervous system with isoniazid is in many cases bound up with the metabolism of pyridoxine and nicotinic acid. The interesting possibility remains that this might be the mechanism in all cases developing neuritis and/or psychosis with isoniazid. It would require further investigation to establish in how many of these cases there was a possibility of a pre-pellagrous condition beforehand, as was the position in the clear-cut case described by Harrison.[21] We note also in this connection that the case described by McConnell and Cheetham[19] was suffering from abdominal tuberculosis—a condition which is known to predispose to pellagra.

As regards the alleged ability of isoniazid to cause insanity *per se*, it is evident that the concept arose from the earliest reports on the effects of the drug. Thus it was reported to have shown evidence of toxicity for the nervous systems of experimental animals[10,11] and later that it caused all manner of excitement, irritability and uncontrollable behaviour in humans, [12,13] and it was stated by the Committee on Therapy of the American Trudean Society[15] that psychosis was found to complicate the administration of isoniazid in some cases and that: 'The psychoses were more common in patients with an unstable personality and those with organic brain disease. They were very serious, in some instances requiring transfer to a mental hospital'. The cases, however, are not described more fully—this was in fact not possible, because the Committee had reported cases quoted by practitioners in answering a questionnaire on the use of isoniazid. The mention of the groups 'unstable personality' and 'organic brain disease' further makes the validity of psychosis as a complication of isoniazid administration questionable.

During the 'early days' a case was also reported in Britain which seemed to indicate that mental disorder might follow isoniazid therapy. Hunter[22] reported that a 41-year-old woman, who had received isoniazid (50 mg. orally 3 times a day for 3 months and double this amount for the ensuing month) presented first with signs of peripheral neuritis and later with a severe acute confusional psychosis with marked fear and visual hallucinosis. Cessation of isoniazid therapy had no notable effect. During the ensuing months her physical condition (as measured by temperature recordings and weight increase) improved and the acute confusional hallucinatory condition cleared, but she lapsed into a chronic state of confusion and disorientation with intellectual impairment, memory defect and emotional lability. The conclusion was drawn that she suffered from cerebral damage caused by the isoniazid *per se*.

This conclusion does not, however, appear to be so clear cut when viewing the evidence today. Further similar cases have not been at all prominent in later literature. On the contrary, various workers have since marvelled at the lack of 'mental symptoms' with this drug and, in fact, at the lack of any form of severe toxicity. Thus the Tuberculosis Chemotherapy Trials Committee of the British Medical Research Council reported in 1953[23] and 1955[24] that they had found no evidence of severe toxicity on doses of 200 mg. of isoniazid per day for periods up to six months. They had worked with hundreds of patients. Similarly, it was reported in America[25] in 1952 that doses of 2-3 mg./kg. per day yielded no evidence of severe toxicity when given for 16 weeks.

In the work by Biehl and Nimitz on 116 cases, already mentioned,[16] peripheral neuritis was fairly common in cases receiving up to 24 mg./kg. per day for periods of more than 4 months, but they were impressed by the absence of 'manifestations which can loosely be grouped under the heading of cerebral or autonomic stimulation'. They conclude 'that a high dosage of isoniazid (20 mg./kg. per day) is well tolerated by the tuberculous patient except for occasional gastric intolerance and for the complication of peripheral neuritis'.

Interesting light is shed on the alleged power of isoniazid to cause mental disturbances by the experiment conducted on 15 patients receiving 100 mg. 3 times per day for 3 weeks.[26] Two objective measurements of emotional status, a clinical evaluation by a trained medical observer, and the patient's own estimation of their mood, were employed. No evidence of any mood changes could be found and the workers concluded that the irritability, elation and excitement reported earlier were due simply to the introduction of a new (and promising) drug for a chronic disease—and that the introduction had enjoyed the accompaniment of considerable 'enthusiasm and fanfare'.

There remains the possibility of precipitating epilepsy. We have mentioned[10,11] that lethal doses of isoniazid in animals caused death by convulsions after signs of cerebral irritation had been evident. EEG studies on anaesthetized dogs[27] also revealed high amplitude spikes with high doses of the drug.

Isolated cases of isoniazid causing epilepsy in humans have been reported. Thus Biehl and Nimitz[16] found an increased seizure frequency in one of their cases who was an epileptic. They remark that pyridoxime might be of use as a prophylactic measure, because it was known to inhibit epilepsy caused by thiosemicarbazide, and it was their conviction that it also prevented neuritis as a complication of isoniazid.

Another case[28] was reported who was known to suffer from seizures of petit mal, but who developed major convulsions on 300 mg. of isoniazid per day. The convulsions stopped when the drug was stopped, but appeared again 4 months after it was started again.

Fetterhoff *et al.*[29] describe a case of a man who had suffered

from post-traumatic epilepsy for 19 years. For this he was receiving phenobarbital (90 mg.) intermittently and Dilantin (0·2 g. orally) 3 times per day. A course of isoniazid (200 mg. per day) was then instituted. On the 1st night he had a seizure and on the 12th night another. He had the last phenobarbital on the 18th day and developed fatal status epilepticus on the 24th day. They conclude that the fatal outcome of this case illustrates the necessity of having cases with epilepsy well controlled with barbiturates before instituting a regime of isoniazid treatment. We might add that such control is necessary in any case of epilepsy, whether he is to receive isoniazid or not, but that the administration of isoniazid will certainly increase the necessity for such control. P'an *et al.*[30] found that such control is likely to be effective in the face of isoniazid administration when they gave small doses of barbiturates to dogs and mice receiving lethal doses of isoniazid.

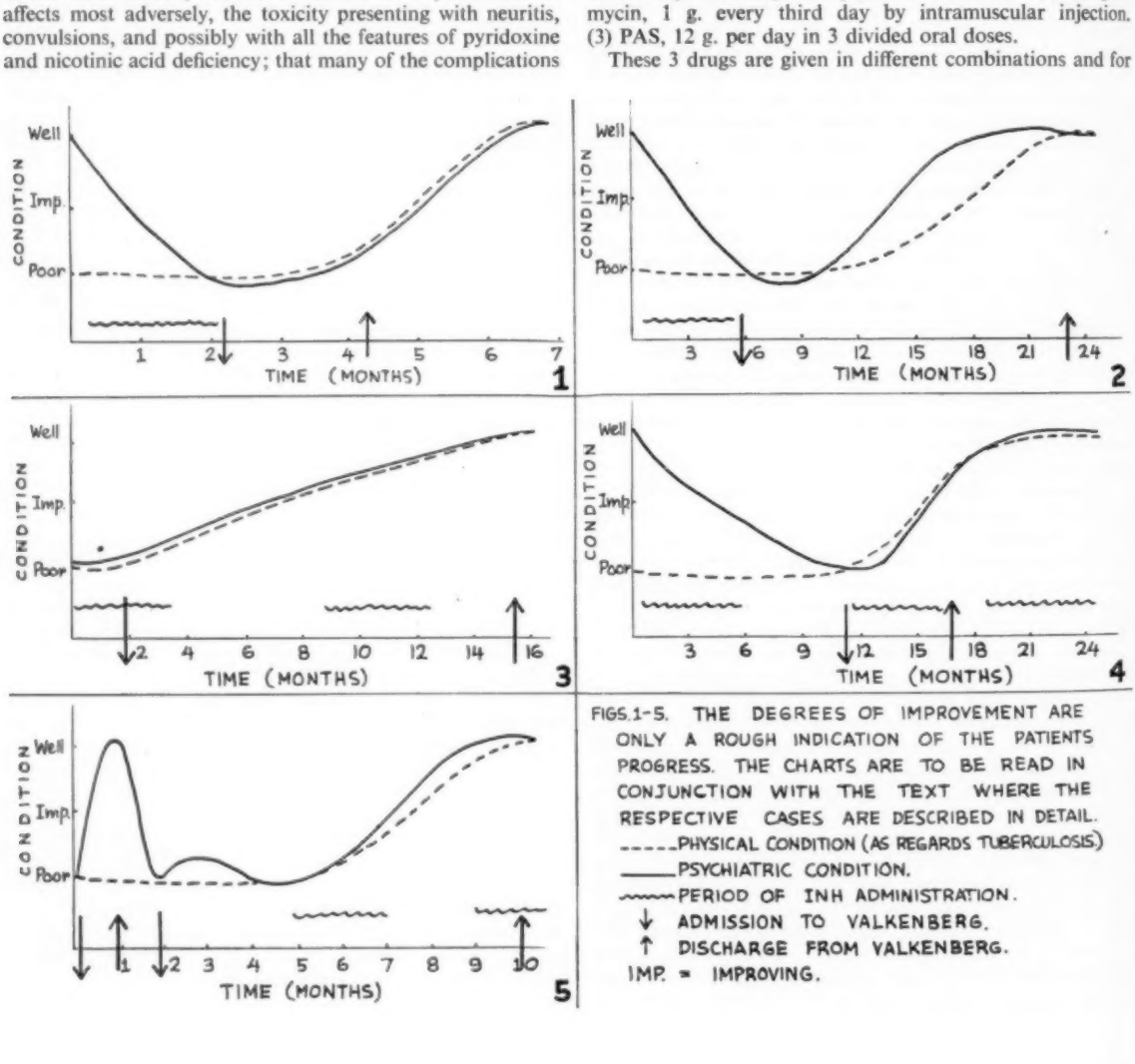
It seems, therefore, that isoniazid is relatively free from severe toxic effects; that it is the nervous system that it affects most adversely, the toxicity presenting with neuritis, convulsions, and possibly with all the features of pyridoxine and nicotinic acid deficiency; that many of the complications can be averted by the prophylactic use of appropriate vitamins and barbiturates; and that the direct causation of mental disturbances and psychoses by isoniazid is doubtful. This is in contrast to the belief still held by many members of the medical profession that isoniazid can, and often does, cause mental disturbance, and that it might be contra-indicated in the presence of any form of mental abnormality.

CASE REPORTS

Routine Treatment Schedule

All cases of tuberculosis in our hospital are isolated in a special ward. They are left more or less to themselves as regards bed rest (enforcement would be impossible when dealing with psychotic people), but they are not employed on the grounds or in the ward, and they receive a special diet. In addition, they receive the following drugs: (1) INH, 300 mg. per day in 3 divided oral doses. This is continued for 56 days and may be repeated if indicated. (2) Streptomycin, 1 g. every third day by intramuscular injection. (3) PAS, 12 g. per day in 3 divided oral doses.

These 3 drugs are given in different combinations and for



FIGS. 1-5. THE DEGREES OF IMPROVEMENT ARE ONLY A ROUGH INDICATION OF THE PATIENTS PROGRESS. THE CHARTS ARE TO BE READ IN CONJUNCTION WITH THE TEXT WHERE THE RESPECTIVE CASES ARE DESCRIBED IN DETAIL.
-----PHYSICAL CONDITION (AS REGARDS TUBERCULOSIS)
——PSYCHIATRIC CONDITION.
~~~~PERIOD OF INH ADMINISTRATION.
↓ ADMISSION TO VALKENBERG.
↑ DISCHARGE FROM VALKENBERG.
IMP. = IMPROVING.
~~~~

varying lengths of time, the regime being decided upon in each case by our advising tuberculosis centre (as indicated above) and regular checks and revisions are made. Charts of treatment schedules and results are carefully kept and annotations made from day to day.

In the description of individual cases, further details of treatment will be mentioned. The individual doses conform to the regime outlined above.

Case No. 1 (Fig. 1)

This was an Indian male aged 17, who had been bed-ridden since February 1955 and was admitted to a general hospital in April. He had a spastic paraplegia and was complaining of root pains across the thorax. The condition was diagnosed as tuberculosis of the spine and a laminectomy was performed at T 3-4 on 3 May. The cartilages were removed and all granulation tissue cleared away.

Ever since the operation he showed a progressive mental disturbance. He was confused, hallucinated and noisy, and was said to suffer from 'a schizophrenic psychosis'. On 8 June 1955 he had to be admitted to Valkenberg. He was still paraplegic, and was wet and dirty, very restless and noisy, overtly hallucinated and confused and disorientated. He also showed various delusions, e.g. that his teacher was bewitching him or that his whole body was blocked with faeces. He was obsessed with the appalling compulsion of constantly scratching faeces from his anus.

This picture was maintained until the end of July, when he gradually started showing less morbid interest in himself and more in his environment. The hallucinations diminished and he became calm and approachable. During August he further improved and could now get around the ward on his own—that is, he could walk unaided—albeit with the aid of beds and chairs. He was now rational in behaviour and speech, orientation was normal and his intimate habits were correct.

He was discharged on leave on 13 August 1955, and recovered completely outside; so that he could be finally discharged before the end of the year.

Treatment. This man was given streptomycin, PAS and INH (details unknown) before his admission to Valkenberg. The INH was discontinued on admission because of its alleged ability to induce mental disorder. Streptomycin and PAS, however, were continued (routine dosage) for the whole of his stay, that is from 8 June to 13 August. No specific psychiatrical treatment had been employed.

Case 2 (Fig. 2)

This Coloured male, aged 29, was a known tuberculotic when admitted to us in October 1954. He had previously received INH, streptomycin and PAS (details unknown) at a tuberculosis clinic in the city from April to September 1954. He had bilateral pulmonary infiltration on admission and fluid at the left base. He weighed 122 lb., his ESR was 13 mm. (Westergren 1 hour) and he was pyrexial.

Mentally he was noisy, restless, overtly hallucinated and disorientated and confused. He needed repeated sedation. This confusional, over-active and irrational picture was maintained for several months and it was not before February 1955 that improvement was noted. The irrational, noisy behaviour was now intermittent only. He was reported to be still better in June and was normal both at interview and in behaviour after July 1955. He was discharged as fit and capable of managing his affairs in January 1956.

Meanwhile his physical condition had likewise improved. His weight increased steadily until September 1955, since when it remained more or less constant at 155-160 lb. On X-ray the disease showed improvement after May 1955 and only calcified foci were reported after August 1955.

Treatment. During his stay at Valkenberg this man, too, was given no INH for fear of its toxicity in a patient already disordered. He was however given PAS and streptomycin (routine dosage) without a break from his admission in October 1954 to his discharge in January 1956. No specific psychiatrical treatment had been employed.

Case 3 (Fig. 3)

When this man, a male Native aged 18, was admitted to Valkenberg in February 1955 he had been treated outside for pulmonary tuberculosis with PAS, streptomycin and INH. The details of therapy were however, unknown.

On admission he was literally carried into the ward. He was in a state of totally incapacitating, acute, confusion; he was overtly hallucinated and resistive to all forms of attention. He was faulty in intimate habits and had to be spoonfed. His general physical condition was so poor that he could not walk.

He remained in this confused, disorientated and hallucinated state for more than a week, during which time he needed every form of nursing care. A fortnight after admission, however, he had improved physically, could sit up and walk and could attend to his basic physiological needs. He was, however, still unable to give any coherent account of himself and speech consisted mostly of irrational answers. He was obviously still hallucinated.

An X-ray taken in March showed bilateral pulmonary tuberculosis. From February until July or August there was gradual improvement both mentally and physically. Step by step he was regaining his intellectual faculties. Speech became progressively more rational and informative. He was, however, still not quite sure of temporal relationships, and he persistently admitted to distressing auditory hallucinations.

His weight had meantime increased from 119 to 150 lb., he was no longer pyrexial, and he was caring for himself in every way and was a willing and effective helper in the ward.

By September another X-ray was taken of his chest and the report was that the right lung had cleared. He was now normal mentally, except for persisting hallucinations.

During October a second course of INH was started, but he remained absolutely well, and was discharged as capable of managing his own affairs in February 1956. He was well enough physically to be treated through an out-patient clinic.

Treatment received. At no stage whatever was any form of psychiatrical treatment employed. He received INH (300 mg./day) for 56 days from the day of admission and again from 11 October for 56 days. He received streptomycin and PAS throughout his stay (routine dosage).

Case 4 (Fig. 4)

This man, a male Native aged 32 years, had been treated as a known case of tuberculosis at an out-patient clinic since September 1954. He had florid, bilateral pulmonary tuberculosis and was treated with streptomycin, PAS and INH (details unknown). During the second half of 1955 (a year after treatment had been instituted) he became psychotic and he was admitted to Valkenberg in August of the same year.

His physical condition was poor on admission. He was pyrexial, and X-rays showed active, bilateral pulmonary infiltration. Mentally he was inaccessible and was belligerent, noisy, overtly hallucinated and disorientated in all fields. This picture of acute confusion continued for about 6 weeks. Towards the end of September he could converse quite rationally, but was still disorientated for time. He was now well behaved and cooperative in the ward.

He improved still further during the following months and no evidence of mental disorder could be established after November of 1955 until his discharge as capable of managing his affairs on 23 January 1956. His physical condition had meanwhile shown similar improvement. Florid bilateral pulmonary infiltration was evident on X-ray up to August 1955, when he was still pyrexial but his weight increased from the end of September, his pyrexia abated, and by December he was constantly apyrexial and showed clearing of the disease on X-rays.

A later communication from an outside tuberculosis centre, dated August 1956, states that he has been so well physically and mentally that he has been working regularly since April. He was then still on regular doses of PAS and INH.

Treatment. This case had received INH, streptomycin and PAS for some months before admission (details unknown). Soon after admission, INH and streptomycin was started (routine dosage). INH was continued for 56 days, streptomycin throughout his stay, and PAS from October until his discharge. It is interesting that he was given INH again after his discharge and was working outside and remaining well for more than 4 months while on INH.

Case 5 (Fig. 5)

A male Native aged 35 years. This man was admitted from a general hospital on 5 April 1955. Why and when he was ad-

mitted there is unknown, but he became so psychotic that he had to be certified. He showed episodes of violence, was totally confused and disorientated, and had obvious auditory and visual hallucinations. He was unable to hold any semblance of a coherent conversation at all.

Towards the end of April, there was spontaneous improvement in this picture and he was described as being normal both in speech and behaviour during May. He was consequently discharged on leave on 11 May 1955.

A month later, however (June 1955) he was readmitted in a state similar to that on his first admission. This time he was given ECT, with some beneficial effect. He became somewhat quieter in behaviour during July but was still confused. By August he was back where he started—irrational in speech and behaviour and hopelessly confused. In September he was found to be pyrexial, and examination of the chest revealed signs of widespread bilateral lesions. X-rays confirmed the diagnosis of pulmonary tuberculosis and routine treatment was started.

The patient remained confused, hallucinated and resistive and during September it was necessary to seclude him because of uncontrolled, impulsive behaviour.

After October, however, he started showing gradual but definite improvement, and all the reports from November until his discharge in February 1956 described him as rational and apsychotic in every way.

The mental improvement was accompanied by physical improvement. His weight showed a steady increase until November and then remained constant. He was apyrexial from November and an X-ray report made in January 1956 mentioned only 'patchy pneumonitis in the right mid-zone'.

Treatment. This man had no special treatment on first admission. Benefit was derived solely from adequate food and rest. Soon after his second admission (June 1955) he was given a course of 15 electro-shock treatments with some temporary effect.

In September 1955 full-scale anti-tuberculous treatment was instituted. He had INH (300 mg. per day) from 11 September to 6 November and again, just before his discharge, from 25 January to 2 February.

Streptomycin and PAS were given in the usual dosages from 7 November until his discharge in February of the next year.

DISCUSSION

In the 1st case described we find, as in all the others, gross mental disorder appearing at the peak of acute physical disease, with the two conditions subsequently showing simultaneous improvement. The improvement in his mental condition, incidentally, took place after the cessation of izoniazid administration. However, in the absence of any other signs of isoniazid toxicity, such as avitaminosis, neuritis or convulsions or, for that matter, elation of mood and excitability, the possibility of INH having caused the mental state is doubtful. This doubt is strengthened by the fact that the mental picture presented was that of the typical toxic confusional state also seen in the other cases, and that it coincided strikingly with improvement in the physical condition.

In the 2nd case also INH was terminated for fear of its toxicity, and again this was followed by improvement of the mental condition, but only 7 months after the drug was stopped. Again, as in the first case, this improvement coincided almost precisely with the physical improvement. A further point of interest here, is that his physical condition remained unimproved before his admission to Valkenberg. This may indicate that his actual drug intake during this period was erratic, as is so often the case with out-patients everywhere. It further casts doubts on the aetiological connection between his INH intake and his mental condition. We note, also, that his psychosis also took the form of an acute toxic confusional state. Again, neuritis and pellagra were entirely absent.

In the 3rd case once again, we find dramatic simultaneous physical and mental improvement, the psychotic condition being that of an acute confusional state. The improvement was gradual and hallucinosis remained for some weeks after all other signs of disorder had cleared (an entirely different picture, for instance, from that of the schizophrenic remission). But this case takes us one step further. We find that the improvement described above took place while the man was actually taking INH. Not only was he on INH during April and March 1955 (when improvement was steadily taking place) but he was also on INH during the last 3 months of the year, when he was taking the final steps to complete mental recovery. There is therefore no doubt that INH was in no way detrimental to this man's mental state. On the contrary, it was a factor in bringing his recovery about inasmuch as it helped to de-activate his tuberculosis.

Once again, in the 4th case, we see a typical confusional type of syndrome and once again there is simultaneous mental and physical improvement—again while the patient was actually receiving INH. After his discharge and while he was working outside, he was again given INH—and he remained well mentally.

As in cases 1, 2 and 3, we also find here that INH administered before his hospitalization had no effect on his physical condition. We must conclude that it is the combined effect of adequate rest and nutrition and regular supervised medication that had brought about both the physical and mental improvement.

In case 5, we find further corroboration for all these conclusions. This man had spent 5 months as a 'mental patient' before his tuberculosis was discovered. During this period he had a short-lived spontaneous remission which resulted in his discharge and almost immediate readmission. This 'remission' might as well have been due to diminished effect of the tuberculous toxicity because of the suddenly improved nutrition and rest provided by hospitalization. Soon afterwards he showed another little peak of improvement—this time simultaneously with a course of electro-shock treatment. The effect was, however, short-lived, and only controlled, regular anti-tuberculous medication (after diagnosis of this condition) brought sustained mental improvement. Once again, this went hand in hand with physical improvement and once again it took place in spite of, and apparently with the aid of INH. He too, had a further course of INH at the end of his stay at Valkenberg and, again, without any deleterious mental effects.

CONCLUSIONS

(*a*) The above small series of cases seems to indicate that tuberculosis *per se* is capable of causing a toxic confusional type of mental disorder, and that the successful treatment of the tuberculosis in these cases results in simultaneous psychiatrical improvement, independently of specific psychiatrical therapy.

In this whole concept there is nothing startlingly new. The findings do, however, underline the necessity of regarding disease as one, regardless of whether it presents with 'organic' or 'mental' features. The consequent importance of adequate physical examination of psychiatrical cases is obvious.

(*b*) Our cases, as well as the bulk of literature on the

subject, indicates that, although isoniazid is notorious for an alleged ability to cause insanity, there appears to be good reason to doubt this allegation in the absence of concomitant vitamin-deficiency. Certainly the drug does not appear to be contra-indicated in cases of mental disorder caused or aggravated by tuberculosis. On the contrary, it seems fair to assume that isoniazid should be employed in these cases, with the following reservations:

Cases of epilepsy should be adequately controlled, preferably with barbiturates.

The possible co-existence of dietary deficiency (especially of the vitamin-B group) should be counteracted.

Signs of peripheral neuritis should be regarded as danger signals, both as regards direct toxicity of the drug, and as regards secondary vitamin-deficiency—notably pellagra. The complete relationship, however, between isoniazid, vitamin deficiency and nervous-system intoxication has not been clearly established.

SUMMARY

1. The literature is reviewed both as regards the psychiatrical concomitants of tuberculosis and the toxicity of isoniazid for the nervous system.

2. Five cases are recorded which suffered from a toxic confusional type of mental disorder concomitant with tuberculosis. In all these cases improvement in the mental state was congruous with that of the physical state.

3. In all 5 cases this dual improvement took place in the absence of any form of psychiatrical therapy, but with the aid of anti-tuberculous treatment. Three of the cases had INH for periods of not less than 2 months.

4. It is concluded that tuberculosis can cause insanity, that the best treatment of these cases is the treatment of the tuberculosis, and that isoniazid is probably not contra-indicated in such cases, but that its judicious use is probably beneficial.

The Commissioner of Mental Hygiene (Dr. I. R. Vermooten) has given consent to the publication of this article, and I wish to express my thanks for this.

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TRAUMATIC DIAPHRAGMATIC HERNIA

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As a result of the increasing incidence of industrial and automobile accidents, as well as of war injuries and assaults, traumatic herniation through the diaphragm occurs more and more frequently nowadays. From being a surgical curiosity it has become common enough to warrant a place in every surgeon's mind, especially when dealing with trauma. Harrington (1948), in a series of 430 cases operated on for diaphragmatic hernia, found trauma to be the causative factor in 13·5% of cases. The following 5 cases seen in the general surgical wards within a period of 1 year, illustrate the clinical features and relative frequency of this condition.

CASE REPORTS

Case 1

A male aged 35 years was injured when he was thrown with considerable force out of a moving motor vehicle onto his abdomen and chest. On admission he complained of shortness of breath and inability to pass urine. He was pale and shocked, with a blood pressure of 100/60 mm. Hg. There was diminished movement of the left chest with dullness to percussion and poor air entry over practically the whole of the left lung. There was tenderness in the epigastrium and left hypochondrium, dullness in both flanks and evidence of free fluid in the peritoneal cavity. Bowel sounds were absent. A haematoma was noted over the left pubic region. A clinical diagnosis was made of fractured ribs with penetration of the lung, ruptured spleen, fracture-dislocation of the pelvis and probable rupture of the posterior urethra.

He was given a transfusion of 500 c.c. of blood. At operation, a thoraco-abdominal incision was made through the left 9th interspace. Practically the whole of the stomach, the transverse colon and about 5 feet of small bowel were found in the left pleural cavity. A large tear, 6 inches in extent, was observed in the left dome of the diaphragm, extending forwards from the crura through the musculo-tendinous junction. In addition, the spleen was ruptured, and about 800 c.c. of blood was removed from the abdomen. Numerous serosal tears were noted in the small bowel and transverse colon, but no actual rupture of bowel

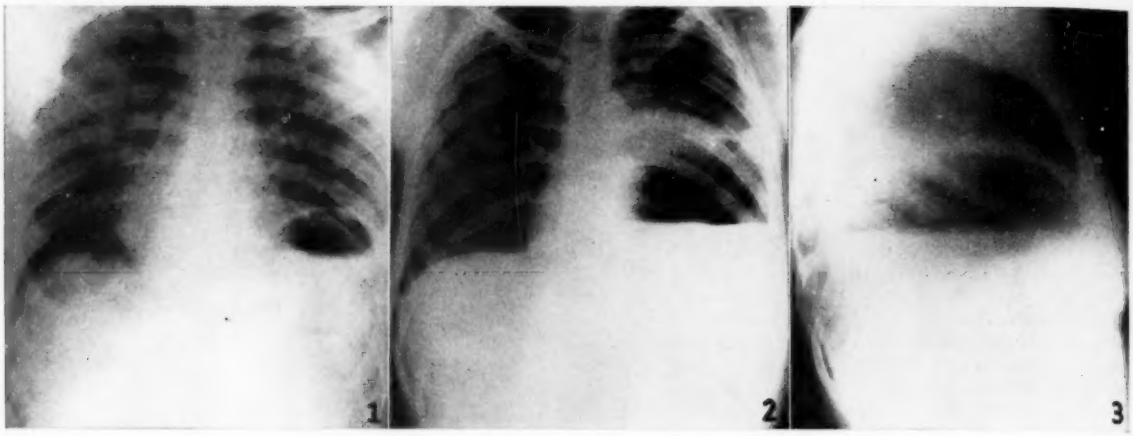

Fig. 1. Case 2. P.A. view of the chest showing herniation of the stomach into the left pleural cavity with a large fluid level in the stomach. Note that this simulates a raised left diaphragm.

Fig. 2. Case 3. P.A. view of the chest showing a large herniation of the stomach into the left pleural cavity with collapse of the left lower lobe and pleural effusion.

Fig. 3. Case 3. Lateral view of the chest showing a large herniation of the stomach with collapse of the left lower lobe.

had occurred. The spleen was removed, the bowel replaced in the abdomen, and the tear in the diaphragm closed with interrupted silk sutures. The chest was closed without drainage. A catheter was passed into the bladder and clear urine obtained.

The following day bowel sounds were present. Radiological examination of the chest revealed adequate lung expansion. Two days later there were signs of consolidation at the left base which resolved on treatment with penicillin and physiotherapy. Ten days post-operatively the patient had fully recovered from his operation. He was however confined to bed for 3 months because of the concomitant pelvic fractures. A barium-meal investigation 6 months later showed no recurrence of the hernia, although the dome of the left diaphragm was slightly elevated.

Case 2

A male aged 23 years was admitted to hospital with a stab wound of the left side of his chest in the 9th interspace. There was dullness to percussion and diminished air entry at the left base. Tenderness and rebound tenderness were elicited in the left hypochondrium. Bowel sounds were normal. The remainder of the examination was essentially negative. Radiological examination of the chest revealed a herniation of the stomach through the left diaphragm (Fig. 1).

A thoraco-abdominal incision was made in the left 9th interspace. Inspection of the left pleural cavity revealed a 2-inch rent in the diaphragm, through which part of the stomach and omentum had herniated. The anterior wall of the stomach had a

Fig. 4. Case 3. Barium-meal investigation in the Trendelenburg position showing a large herniation of the stomach into the left pleural cavity with constriction at the diaphragm. Note the colonic shadow above the stomach.

Fig. 5. Case 5. P.A. view of the chest showing varying densities and shadows at the left base.

Fig. 6. Case 5. Barium-meal investigation in the Trendelenburg position showing herniation of the stomach and colon into the left pleural cavity.

non-penetrating laceration, which was oversewn. The abdominal contents were replaced and the rent in the diaphragm repaired with interrupted silk sutures. About 300 c.c. of blood was aspirated from the pleural cavity. An intercostal drain was inserted and the chest and abdomen closed.

The following day the lung had completely re-expanded, and the intercostal drain was then removed. The patient made an uneventful recovery and was discharged from hospital 14 days after admission.

Case 3

A 28-year-old female was admitted to hospital with a stab wound in the left side of the chest. She was in a state of shock, with a blood pressure of 90/40 mm. Hg. Surgical emphysema was present over the left chest. Air entery was adequate in both lungs. No adventitious sounds were heard. The remainder of the physical examination was negative. She was treated with intravenous fluids, penicillin and sedation, and her condition improved considerably. Radiological examination of the chest was very suggestive of a diaphragmatic hernia (Figs. 2 and 3). Barium-meal studies revealed the greater part of the stomach to be above the diaphragm. In addition a gas shadow was visible above the stomach, probably due to colon (Fig. 4).

The patient felt very well, refused operation and went home. Two weeks later she returned, complaining of vomiting after meals and upper abdominal pain which was relieved by sitting or standing up. She now consented to operation.

Thoracotomy was performed through the left 8th intercostal space, and inspection revealed collapse of the left lower lobe and a 2½-inch rent in the left dome of the diaphragm through which a large part of the stomach and splenic flexure had herniated. These were bound down by numerous adhesions, presumably due to a perforation which had sealed off. After the adhesions had been freed, the stomach and colon were returned to the abdomen. The rent in the diaphragm was repaired with interrupted silk sutures. The left lung was inflated until it had fully re-expanded and the chest was then closed without drainage.

Post-operatively the patient developed a remittent temperature ranging between 99 and 103·5°F which did not respond to antibiotics. Three weeks later a rib resection was performed for drainage of an empyema, which healed within 3 months. Barium-meal studies subsequently showed that there was no recurrence of the hernia and the diaphragm moved well.

Case 4

A 43-year-old male was admitted to hospital complaining of severe pain in the chest, shortness of breath and vomiting of 2 days' duration. He further stated that 3 months previously he had been stabbed in the left chest, and had been admitted to another hospital, where a laparotomy had been performed. He was however symptom-free until 2 days before this admission.

The patient was seen to be extremely ill, anaemic and dyspnoeic, and in obvious pain. His blood pressure was 110/60 mm. Hg. There was pain and tenderness in the upper abdomen, and a left upper abdominal laparotomy-scar was noted. There was dullness on percussion, poor air entry and diminished vocal fremitus over most of the left chest, with a shift of the mediastinum to the right. On radiological examination an opacity was seen in the lower half of the left chest and a diagnosis of pleural effusion was made. Blood-stained fluid was aspirated from the chest. Repeat X-ray revealed a large gas shadow in the midst of this opacity, and the condition was now diagnosed as herniation of the stomach through the diaphragm, with strangulation.

After the patient had received a transfusion of 500 c.c. of blood, the abdomen was explored through an upper transverse abdominal incision. A large rent was observed in the left diaphragm through which the stomach had herniated. The incision was extended into the left chest through the 8th interspace and it was then seen that the stomach, which was lying in the left pleural cavity, was oedematous and plum-coloured. About 500 c.c. of blood-stained fluid was aspirated from the pleural cavity. The blood pressure had dropped to 60/40 mm. Hg and the patient's condition caused great anxiety. There was some doubt about the viability of the stomach, but in view of the patient's extremely grave condition, the adhesions were freed and the stomach returned to the abdomen. The diaphragm was then closed with interrupted silk sutures. With resuscitative measures his condition improved. Two days later he developed a gastric fistula, and on the 3rd day the abdominal wound broke down, revealing that most of the stomach, except for the posterior wall and fundus, was gangrenous and sloughing. A catheter was inserted into the duodenum and another into the lower end of the oesophagus. Both were brought out through the abdominal wound. The abdomen was then resutured. In view of the patient's poor condition, no further surgery was attempted, but 36 hours later the wound had burst again, and in spite of intravenous fluids, antibiotics and noradrenalin infusions, the patient's condition deteriorated and he died of peritonitis on the 5th post-operative day.

Case 5

A male aged 23 years was admitted to hospital. He stated that 3 hours previously he had been involved in a motor accident, when his chest had collided with the steering wheel of the car which he was driving. He complained of pain in the epigastrium, left chest and the left shoulder. The chest pain was aggravated by deep breathing.

His blood pressure was 120/60 mm. Hg, pulse rate 88 per minute and he was slightly cyanosed. The trachea was deviated to the right. At the left base there was dullness to percussion, diminished air entry and diminished vocal fremitus and resonance. No adventitious sounds were heard. Tenderness and rigidity were elicited in the left upper abdomen. Bowel sounds were present.

Radiological examination revealed a raised left diaphragm, with gas shadows and densities in the left pleural cavity suggestive of diaphragmatic herniation (Fig. 5). Barium-meal studies demonstrated a herniation into the chest of the whole stomach and a loop of colon (Fig. 6). It was interesting to note that the ingestion of barium was only possible with the patient in the sitting position or when lying on his right side. Any other position caused vomiting.

The chest was opened through an incision in the left 8th intercostal space. The left lung was found to be partially collapsed, and a rent 5 inches long was detected in the diaphragm. This commenced at the oesophageal hiatus and extended towards the dome. The stomach, large bowel, small bowel, spleen and omentum had herniated through this tear. The viscera were returned to the abdomen, the diaphragm was repaired with interrupted sutures, and the chest was closed without drainage. Except for a slight pleural effusion, which did not require any active procedure, the patient made an uninterrupted recovery and was discharged from hospital 10 days after admission.

DISCUSSION

The commonest causes of traumatic diaphragmatic hernia are direct and indirect violence. It has also been known to arise as a result of a subdiaphragmatic abscess with necrosis of the diaphragm, erosion from a drainage tube for empyema of the chest, and post-operative dehiscence of a diaphragmatic wound.

Herniation caused by indirect violence is usually due to a sudden compression of the upper abdomen and lower chest, such as is sustained when the chest is crushed by the steering wheel of a car (case 5), or in falling from some height onto the chest wall (case 1). These injuries are often associated with fractures of ribs, and less frequently with rupture of the spleen. The resulting tear commonly begins near the oesophageal hiatus and radiates peripherally to the dome of the diaphragm or through the musculo-tendinous junction. It may however extend to the central tendon. The tear is usually large and allows the herniation of large amounts of abdominal contents into the pleural cavity. Negative pressure in the thorax combined with positive abdominal pressure 'sucks up' the stomach with or without omentum, transverse colon, small bowel and spleen into the chest in descending order of frequency, as is well illustrated in cases 1 and 5.

Direct violence is generally due to gun-shot wounds or stab wounds (cases 2, 3 and 4). The site and size of the

defect will depend on the type of instrument used, but the resulting hernia is usually smaller than in herniae due to indirect violence. Initially, there may be minimal herniation or none at all. This is due either to the small size of the rent in the diaphragm or to plugging of the rent by omentum, spleen or liver. Any relatively sudden increase in abdominal pressure at a later date may cause an increase in the size of the hernia and aggravate the symptoms. Radiological examination may only then become positive (Schneider, 1956). Plugging of the rent by a large organ like the liver probably accounts for the rarity of right-sided lesions. Traumatic herniation through the right leaf occurs in only 2 - 5% of cases (Carter *et al.*, 1951).

The symptoms and signs of traumatic diaphragmatic hernia may be conveniently divided into 3 phases: an initial phase, an interval phase and an obstructive phase.

In the *initial phase*, the symptoms and signs of the hernia are not infrequently overshadowed or disguised by concomitant lesions, e.g., in case 1, where the presenting features were those of fractured pelvis and ruptured spleen. Pain in the left upper abdomen and lower chest, which may radiate to the shoulders, is a fairly constant feature. On the other hand, the patient may only complain of mild abdominal discomfort. The signs of the hernia are mainly referable to the cardio-respiratory system, viz. diminished excursion of the thorax, dullness to percussion at the left base (but tympany may be present), diminished air entry, adventitious sounds, succussion splash, mediastinal shift to the unaffected side, and cyanosis and dyspnoea which may go on to circulatory collapse.

The *interval period* varies from a few hours to many years and usually presents with vague symptoms of dyspepsia, post prandial vomiting and discomfort in the epigastric region, and is often diagnosed as gall-bladder disease, peptic ulceration or coronary thrombosis. Therefore, vague gastro-intestinal symptoms together with a recent history of injury to the upper abdomen or lower chest should arouse suspicion of a traumatic diaphragmatic hernia. In this phase, however, it is often discovered accidentally.

The *obstructive phase* is often precipitated by a relatively minor injury or episode causing a raised intra-abdominal pressure. The onset is usually sudden, with severe pain in the left hypochondrium and thorax, which may radiate to the neck and shoulders. Involvement of the colon results in large-bowel obstruction with generalized abdominal pain. The abdomen is then distended, tympanitic and hyperperistaltic. When the stomach is involved, vomiting is an early feature; the vomitus at first consisting of clear gastric contents without bile, later proceeding to frank haematemesis. The latter may be an ominous sign of strangulation. Involvement of the stomach often results in a scaphoid abdomen with normal bowel-sounds. In addition, chest symptoms and signs similar to that found in the initial phase may be present. Pleural effusion is not uncommon and varies from a few hundred to a few thousand c.c., initially straw-coloured but rapidly becoming bloody with the onset of strangulation.

The above sequence of events is well illustrated in case 4. After receiving a stab wound in the chest, the patient was symptom-free for 3 months, followed by the sudden onset of severe pain and vomiting, proceeding to strangulation of the stomach.

In addition to strangulation, other complications of traumatic diaphragmatic hernia are haematemesis, melaena, perforation of a viscus, empyema and thoraco-gastric fistula.

Radiological examination is the most useful investigation in the diagnosis of traumatic diaphragmatic hernia. P.A. views of the chest in the erect position will often suggest the presence of a hernia. The radiological features of this condition are:

(*a*) An abnormally high left diaphragm.

(*b*) Fluid levels in the chest (actually in the viscus herniated through the diaphragmatic rent).

(*c*) Shift of the mediastinum to the opposite side.

(*d*) Areas of atelectasis above the diaphragm.

(*e*) Pleural effusion.

(*f*) Large-bowel or small-bowel patterns in the left thorax.

(*g*) Unusual shadows above the diaphragm, viz. abnormal densities interspersed with gas shadows. Note that the wall of a herniated viscus may often simulate a raised diaphragm.

(*h*) Screening of the chest may show absent or diminished movement of the supposed diaphragm.

Radiologically the diagnosis must be differentiated from a pyo-pneumothorax, encysted pleural effusion, eventration of the diaphragm, hiatus hernia and subphrenic abscess. Should any doubt exist, a barium meal and/or enema examination will confirm the diagnosis. Herniation of the stomach may result in a partial or complete constriction at the diaphragmatic rent, in which case a barium meal will reveal a narrowing or total obstruction at this point. The stomach may or may not fill with barium, depending on the site of the obstruction. Similarly, barium-enema investigation may reveal obstruction in the colon.

The treatment of traumatic diaphragmatic herniae is always surgical, and should be carried out as soon as possible; it has been shown that the mortality is at least doubled with the onset of obstruction (Hedblom, 1951). Pre-operatively, an attempt should be made to correct any fluid or electrolyte imbalance. It should be stressed that the introduction of a Ryle or Levine tube into the stomach of a case of gastric herniation may be a life-saving measure, as the aspiration of a few thousand c.c. of fluid plus a considerable quantity of gas may convert a moribund patient into a fairly good operative and anaesthetic risk.

The surgical approach is preferably transthoracic. If this exposure is inadequate, or reduction of the hernia proves difficult, or if injury to the abdominal viscera is suspected, the incision may be extended into the abdomen, either obliquely or as a transverse upper abdominal incision. Approximation of the diaphragmatic wound edges is usually performed without difficulty. Should any difficulty arise, however, as in an old-standing lesion, one may have to resort to phrenic crush.

Strangulation is a not uncommon complication of traumatic diaphragmatic hernia. 90% of strangulated diaphragmatic herniae are traumatic in origin (Carter, *et al.*, 1951). This is in contrast to hiatus hernia, where strangulation is infrequent. The hazard of this complication increases markedly with any delay in surgical intervention. It is therefore necessary to bear in mind the entity of traumatic diaphragmatic herniation in any upper abdominal and lower chest injuries, and to institute adequate surgical procedures with the utmost expediency.

SUMMARY

1. The clinical features of 5 cases of traumatic diaphragmatic hernia are presented.
2. Significant features in the diagnosis are discussed.
3. The necessity for early diagnosis and surgical intervention is stressed.

I wish to thank the Superintendents of Baragwanath and Johannesburg Hospitals for permission to publish these cases.

My thanks are also due to Messrs. S. Kleinot, R. L. Almond and L. Stein for their assistance and guidance.

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A CASE OF PULMONARY TUBERCULOSIS OF BOVINE ORIGIN IN MAN IN SOUTH AFRICA

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On 26 April 1956 a European male aged 46 years was admitted to King George V Hospital with a history of lethargy, cough and haemoptysis. He had lost 30 lb. in weight during the previous 3 months.

A radiograph of the chest (Fig. 1) showed a tuberculous cavity 5 cm. in diameter situated in apex of the left lower lobe and surrounded by patchy infiltration involving both upper and lower lobes. Minimal pleural thickening was evident in the right middle zone. The first sputum examined was positive for-acid fast bacilli on direct examination.

The patient was treated with daily isoniazid, 20 mg. per kg. body-weight, streptomycin, 1 g. daily for 4 months, and potassium iodide 30 gr. daily for 1 month. The foot of his bed was raised about 12 inches and this posture maintained while he was in hospital. He improved steadily. His sputum converted in approximately 3 months. He gained 40 lb. in weight in 6 months, and on discharge from hospital on 30 November 1956 a radiograph of the chest (Fig. 2) showed residual fibrosis in the left middle and upper zones. Since then he has remained well on 800 mg. of isoniazid daily.

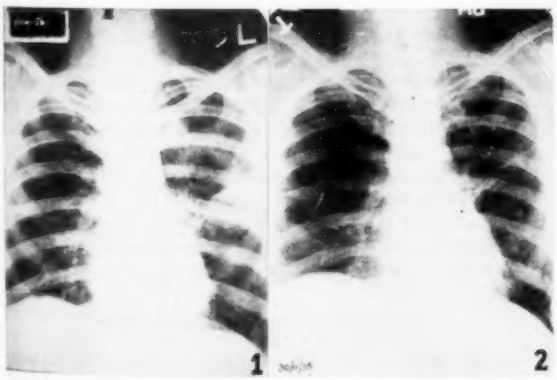

Fig. 1. On admission, 27 April 1956.
Fig. 2. On discharge, 30 November 1956.

Identification of M. tuberculosis, Bovine Strain

On 8 June 1956 a culture of the initial sputum was brought for further investigation. It was seeded on non-glycerinated Lowenstein-Jensen medium, and displayed numerous pale, small, acid-fast colonies, while the tube of the same medium containing glycerin showed no growth after 38 days' incubation. The culture was transferred immediately to du Bos liquid medium and other selective media. The cultural features were studied; they showed dysgonic growth and typical behaviour of *M. tuberculosis*, bovine strain.

A young rabbit was injected into the abdominal flank region subcutaneously, with 0·5 c.c. of a 7-days-old du Bos culture. The animal lost weight rapidly, and 25 days after injection showed distressed breathing and was *in extremis* when it was killed. Autopsy revealed extreme emaciation and a subcutaneous abscess involving the abdominal muscles at the site of injection. The regional lymph-nodes showed caseation. Small tuberculous foci were noted on the peritoneal surface of the colon. The spleen was enlarged and showed numerous pin-point tubercles. Both kidneys showed the presence of several fair-sized sub-capsular tubercles. The liver was slightly enlarged and showed a few small scattered tubercles. The lungs were voluminous in size, filling practically the whole thoracic cavity, and were studded with numerous miliary tubercles. Direct smear examination from the lung tissue showed the presence of many acid-fast bacilli slightly thicker and shorter than when human strains of *M. tuberculosis* are observed in animal lesions.

On 24 July 1956, a second, more mature, rabbit was injected intravenously from a third generation of the same strain. The rabbit weighed 2,012 g. and was given 0·1 c.c. of a 7-day du Bos growth in the marginal vein of the ear.

On 12 September 1956 (49 days after injection) the dying animal was chloroformed and found to have lost 537 g. in weight. Autopsy revealed marked emaciation; the lungs were voluminous and studded with numerous miliary tubercles

similar to those in the previous animal. The enlarged spleen displayed several tubercles. The liver was enlarged, but no tubercles were seen. There were tubercles on the peritoneum of the large intestine and in the perirenal area. A few raised tubercles were visible under the capsule of the kidneys.

A single colony cultivated from a second sputum of the same patient on non-glycerinated Lowenstein-Jensen medium was transferred to du Bos medium. It was well shaken, and grown for 6 days, and then 0·1 c.c. of the liquid growth was injected into the ear vein of a rabbit.

Fifty five days after injection, the dying rabbit was killed; it was found to have lost 420 g. in weight. Again, the lungs were much enlarged, filling practically the whole thoracic cavity. Both lungs were studded with tubercles. The liver was enlarged and showed patchy grey tuberculous areas. The enlarged spleen displayed many caseating tubercles of

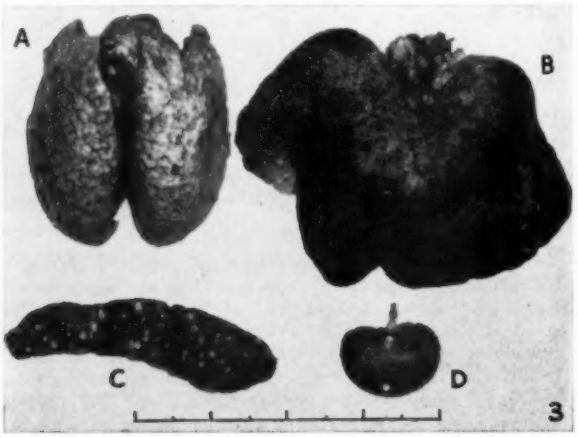

Fig. 3. Photograph showing tuberculous foci produced by bovine strain of human origin, in rabbit which died 55 days after infection. Organs fixed in formalin. Lesions are extensive on the greatly enlarged lungs. A=lungs, B=liver, C=spleen, D=kidney. Scale in inches.

varying size. A few mesenteric glands were enlarged. Under the capsule of the kidneys a few raised tubercles were prominent. Smears from the diseased lung and spleen showed acid-fast organisms morphologically similar to those observed in the lesions at the first rabbit autopsy.

SOURCE OF INFECTION

The patient came from outside the Durban municipal area. He informed us that before his illness he had been drinking raw milk from a dairy near his home. Meanwhile, in the same township, a case of tuberculous cervical adenitis in a European boy of 12 years was discovered by the Medical Officer of Health, who promptly started investigations. It was found that this boy was consuming raw milk from the same dairy. The Government Veterinary Department was notified. This led to tuberculin testing of the herd, which showed a high degree of infectivity.

GOVERNMENT VETERINARY REPORT

On 9 October 1956 Dr. A. I. Robertson, private veterinary practitioner, and one of us (W.B.H.) carried out a single intradermal tuberculin test on 34 cows and 1 bull, these comprising the whole herd. The tuberculin used was human type P.P.D. 10 T.U.

The test was read on 12 October and showed 23 positive reactors, 7 suspicious cases and 5 negative cases. Only 3 or 4 of the animals appeared to be in low condition, with one cow showing a persistent cough. The remainder of the animals appeared, at a glance, to be in reasonable physical condition.

Four of the positive reactors, chosen at random, were slaughtered at the Durban municipal abattoir on 17 October, and all showed caseous lesions in one or more of the following lymph glands: retropharyngeal, bronchial and mediastinal.

After the slaughter of these four cows the owner was informed that, judging from the widespread infection in the herd, it was possible that one or more of the animals might be an open pulmonary case, and that some of the negative reactors might show tuberculous lesions at autopsy, such animals possibly showing tuberculous anergy. Eventually the owner decided to send the whole herd to the Durban municipal abattoir for slaughter.

On 19 October one of the positive reactors died, and the manager later reported the presence of nodules on the pleura and throughout the lung substance. The remaining cattle were slaughtered at the Durban municipal abattoir on 6 November. Of these, 19 animals showed tuberculous lesions varying in degree from localized glandular involvement to miliary and cavernous pulmonary cases. Dr. M. C. Lambrechts, Senior State Veterinarian, Tuberculosis Control, Pretoria, was present at the slaughter of the final 30 animals.

Tuberculin Testing of Human Contacts

Following these findings, available human contacts were tuberculin tested (10 T.U.). The results are shown in Table I.

TABLE I. BOVINE TUBERCULOSIS CONTACTS IN A PERI-URBAN TOWNSHIP X-RAYED AND TUBERCULIN TESTED

Age-group	*Total*	*Mantoux Negative*	*Mantoux Positive*	*D.N.A.*
Infants	2	2	—	—
1– 5 years ..	17	17	—	—
6–10 years ..	19	17	—	2*
11–15 years ..	7	6	1	—
16 and over ..	61	20	35	6*
	106	62	36	8

* D.N.A.=did not attend for reading.

All chest films were normal on mass miniature radiography. It should be noted that the total of 106 persons is made up partly of direct contacts, but all had been supplied with milk from the dairy. Only 1 out of 45 children under the age of 16 years was a positive reactor. No information was obtained whether these children were in the habit of taking their milk raw or boiled. Nevertheless, it is of interest to speculate on the reason for this low degree of infection as shown by the tuberculin test.

RECORDED CASES IN SOUTH AFRICA

As far as we know, this is the first recorded case of pulmonary tuberculosis of bovine origin found in South Africa in a hospital patient and traced directly back to the source of infection.

In contrast to the wide distribution of tuberculosis in dairy herds in the Union, recorded authentic cases of bovine origin in man are infrequent.

Pirie,[1] in 1932, examined 100 mine workers and 98 cases of bone, glandular and meningeal tuberculosis in the Union. No bovine cases were encountered.

Harington and Emerson,[2] in 1939 examined 100 cases of human tuberculosis in Port Elizabeth. Of these, one bovine strain was recovered from the stool of a child.

Du Toit and Buchanan,[3] in 1942, isolated and identified a bovine strain from the cerebrospinal fluid of a young European female.

In 1953, out of 200 cases of human tuberculous meningitis examined in the Western Province, Coetzee[4] recovered strains of bovine origin from 2 cases.

In 1954, Buchanan,[5] working on the Witwatersrand, examined 266 selected non-pulmonary human cases of tuberculosis, culturally and biologically; he found that 13 were infected with the bovine strain.

COMMENT ON LOCAL CASE

It is possible that initially extreme non-glycerophylic bovine strains in man might be missed, as when cultures are made on glycerinated media only, or where the tube containing no glycerin has become contaminated, or where animal inoculation has not been attempted.

Points of interest regarding the original culture in this case were, firstly, the great number of viable organisms as shown by numerous colonies grown from a sputum collected 4 days after the patient's admission. Secondly, that although subsequent sputa showed the presence of acid-fast organisms over a long period, they did not prove viable in bacteriological and animal-inoculation tests.

Cultures made 4 days after admission to hospital gave numerous colonies along the slant of inoculation.

Cultures made 83 days after admission, on 3 glycerinated and 3 non-glycerinated tubes, produced only one visible colony on one non-glycerinated medium.

Eighty-eight days after admission one specimen of sputum was still positive, showing groups of short, irregular acid-fast bacilli. Cultures made, and animals inoculated from this specimen gave negative results.

After this date the sputa became normal. Two gastric lavages and 2 sputa taken at different times proved negative on direct examination, culturally and biologically, before the patient was discharged.

We can only conclude, that the non-viable acid-fast organisms still found at one stage of this investigation were due to the effect of chemotherapeutic substances on the bovine tubercle bacillus and perhaps to some other unknown factor. Like the first, the second culture remained very sensitive to isoniazid.

IMPORTANCE OF PASTEURIZATION

It has been stated by Dr. G. D. English, Medical Officer of Health, Durban, that 97% of the city's milk supply is pasteurized; the balance is well controlled by the veterinary and inspectional staff of the Health Department. There is, therefore, little danger from this the main source of bovine tuberculosis infection in man. This is confirmed by the negative results obtained in the typing of over 1,000 tuberculous cases, mostly pulmonary, at King George V Hospital.

Public health authorities often ascribe the changing pattern of tuberculosis to modern treatment, but pasteurization must be regarded as an important factor.

The occurrence of bovine tuberculosis in cattle is out of proportion to the incidence of the disease in man. In 1930, dairy herds supplying milk to Durban were tuberculin tested by the Government Veterinary Department. This resulted in the slaughter of many infected cattle and potential spreaders of the disease. After the alarming results of this trial test, the City Council embarked on a progressive pasteurization policy. Natal, perhaps as a result of past experience, is more 'pasteurization conscious' than most other parts of the Union.

Although a valuable adjunct to public health, pasteurization should not be regarded as the main defence against milk-borne tubercle bacilli. Total eradication of bovine tuberculosis in cattle should be a national objective. In America, a progressive fall in positive tuberculin reactors in man has been noted since the bovine tuberculosis eradication scheme was inaugurated. The need for a uniform system of milk control for all peri-urban areas in this country is evident, with special reference to the enforcement of regular tuberculin testing of cows producing raw milk for public consumption. At present an animal with a positive tuberculin test is given the Government brand and its disposal is at the owner's discretion; i.e. he may retain the animal, have it slaughtered, or sell it at a public auction.

Since the Bantu race suffers more from tuberculosis than Europeans in South Africa, it is surprising that the first case of bovine pulmonary tuberculosis in man recorded in the province of Natal should be that of a European. This may be due to the fact that Natives in rural areas and in their home environment have a preference for sour milk. It has been shown by Dormer *et al*,[6] that calabash milk or *amasi* has a destructive effect on tubercle bacilli.

SUMMARY

A case of pulmonary tuberculosis in man caused by a bovine strain, and the tracing of the infection to its source, is recorded.

We thank Dr. B. A. Dormer, Medical Superintendent, King George V Hospital, for personal interest taken in this work; Dr. A. M. Beemer and laboratory staff for their assistance; Dr. H. G. H. Houghton for kind cooperation; Dr. M. H. J. van Rensburg for carrying out the tuberculin test on human contacts and Dr. C. C. Wessels, Director of the Municipal Abbattoir, Durban, for reporting on slaughtered herd and Mr. D. Mostert for photographs.

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UNUSUALLY EARLY MANIFESTATIONS OF PORPHYRIA CUTANEA TARDA

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In recent years it has become apparent that porphyria is not a rare disease in South Africa. Most of the cases seen in white members of the community are familial and present the features of skin sensitivity in adult life. Cutaneous manifestations predominate in males and acute episodes are infrequent; in females acute attacks are more common and cutaneous lesions are less severe, sometimes even absent. The children of affected parents have not shown clinical manifestations. This form of porphyria almost certainly falls into the hepatic group defined by Watson (1951), though this has not yet been proved by liver biopsy. Two cases of congenital (erythropoietic) porphyria have been found in Bantu female children not known to be related, but no case has yet been found in a White child.

The case to be described shows a marked resemblance clinically to the congenital form of the disease (see Figs. 2-5), but full investigation has shown that most probably he is a familial hepatic porphyric. The early onset is an exception to previous experience of this disease in South Africa.

CASE REPORT

J.J.C., a White male aged 5 years, was referred by Dr. Deacon to the Out-patient Department of the Krugersdorp Hospital on 29 December 1953 with a history of skin eruptions from the age of 6 months on his face, hands, arms and legs. He had suffered convulsive attacks in his first 5 months but these did not recur after the onset of the skin lesions. When first seen he had vesicles, haemorrhagic bullae, impetiginized areas and marked scarring of the exposed skin, the distal phalanges of the fingers were contracted, and the finger nails were dystrophic. Hirsutism was noted on the temples but his teeth were normal in appearance. He was admitted to the children's ward for observation with the diagnosis of epidermolysis bullosa congenita.

A blood count on 31 December showed 13·0 g.% of haemoglobin and 4·48 million red cells per c.mm., which were normal in appearance. Serum albumin was 3·2 and globulin 4·6 g. per 100 ml. The Kolmer cardiolipin W.R. was negative. Porphyric disease was considered, and a specimen of urine on 27 January 1954 showed a very faint trace of porphyrin on spectroscopic examination. Two more specimens were sent for confirmation; neither contained more than a trace of porphyrin, which was shown to be largely coproporphyrin. On these grounds the possibilities of liver disease or increased haemopoiesis were suggested as explanations for the porphyrinuria. The abnormal serum proteins favoured the former. Haemoglobin on 9 February was 11·6 g. per 100 ml. and liver function tests gave the following results: Thymol turbidity 4·0 units, thymol flocculation 2 plus, colloidal red 3 plus, cephalin-cholesterol 4 plus (24 hours), Takata-Ara 1 plus, zinc sulphate 30 units. Total lipids, alkaline phosphatase, van den Bergh reaction and bilirubin content were all normal. Serum proteins were: albumin 3·4, total globulin 4·3 and gamma globulin 2·2 g. per 100 ml.

These findings appeared to substantiate the suggestion of liver dysfunction and the child was taken from hospital. The mother stated later that he was seen by several other doctors whose treatments, including protection from sunshine, had no effect on the condition. Towards the end of 1955 she took him to the out-patient department of Edenvale Hospital, where congenital porphyria was considered, but a specimen of urine was reported negative for porphyrin. She did not comply with an invitation to return later to see the consulting dermatologist, but a description of the case was given to one of us who obtained their address and visited the home. The history above was repeated and in addition it was learned that though usually normal in colour the urine was sometimes as dark as black coffee. The mother also replied that similar lesions were not known in other members of the family. A specimen of urine obtained at this visit was slightly darker than normal and showed quite unmistakably the presence of porphyrin on spectroscopic examination; porphobilinogen was not detected.

Arrangements were then made for the child to be admitted to the Transvaal Memorial Hospital for Children in Johannesburg for further study. This was done on 10 February 1956. The child, now aged 7, was found to be mentally retarded. Height 49½ inches, weight 47½ lb., pulse rate 100-130 per minute, average respiration rate 28 per minute, and temperature normal. The liver was considered to be within normal limits. No abnormal findings other than the state of the skin were discovered. X-ray examination disclosed no significant abnormality in the long bones or chest. The terminal phalanges of the fingers showed marked resorption (Fig. 1). This was noted in a case report by Garrod (1936).

Unexposed portions of the body showed symmetrical areas of hyperpigmented skin, particularly on the flanks and lower abdomen. Their distribution was segmental with scattered hyperpigmented plaques and *café-au-lait* areas suggestive of von Recklinghausen's disease.

Scalp—scattered scars but no active lesions.

Face and forehead—scarred, with denuded crusted patches on cheeks, forehead and the bridge of the nose (Fig. 2).

Lips—drawn in, with rhagades at the angles giving an appearance of scleroderma.

Neck—active crusted lesions on the back of the neck with hyperpigmentation and slight scarring.

Upper arms—hyperpigmented and a few crusted lesions.

Lower arms—pigmented scars on flexor aspects.

Hands—denuded and crusted impetiginous lesions and scarring of the dorsal surfaces. Fingers contracted and some nails destroyed, particularly the thumb-nails (Fig. 4).

Knees and lower legs—scarring, bullae and haemorrhagic crusted areas (Fig. 3).

Feet—numerous scattered pigmented scars on the lateral aspects of both feet.

Hirsutism was marked on the face, arms and legs.

His teeth were white in daylight and showed no fluorescence when examined in Wood's light.

The following laboratory results were reported on 14 February: Thymol turbidity 6·5 units, thymol flocculation 4 plus, colloidal red 4 plus, cephalin cholesterol 2 plus, Takata-Ara 3 plus, zinc sulphate 45 units. Total lipid, alkaline phosphatase, van den Bergh and bilirubin concentration normal. Serum albumin 2·4, total globulin 5·8 and gamma globulin 3·05 g. per 100 ml. Haemoglobin 8·3 g. Erythrocytes 3·35 million. Haematocrit 26.

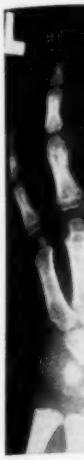

Fig. 1. X-ra
langes.

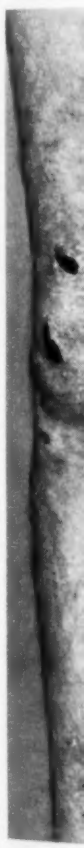

Fig. 3. Legs

Fig. 1. X-ray of hands showing resorption of terminal phalanges.

Fig. 2. Face showing crusted ulcers and hypertrichosis.

Fig. 3. Legs showing crusts and scars.

Fig. 4. Hands and forearms showing crusts, scars, and contraction of fingers.

M.C.H.C. 32. The modified Ide test was negative. Films of bone marrow did not show fluorescent normoblasts.

Whilst in hospital his urine became darker and a 24-hour specimen contained 252 μg. of uroporphyrin and 660 μg. of coproporphyrin. Two stools were analysed and found to contain coproporphyrin 660 and 840, and protoporphyrin 810 and 1,300 μg. per g. dry weight respectively.

With these results in hand the parents were interviewed and it was explained to them that the findings were against congenital porphyria and that there was no reason to expect that splenectomy would relieve the skin condition. The question of a familial condition was again raised and the father remarked that his wife's skin was fragile. She then agreed that this had been the case for the past 10 years at least but that, though ulcers formed easily, they did not as a rule leave visible scars. She had then a small slow-healing ulcer on one knuckle which had resulted from a slight scratch from a rose thorn, and an old pigmented scar on her left ankle. There were no other scars or skin lesions apart from a few moles on her face, neck, shoulders and back, nor was there any hypertrichosis. She had no history of abdominal pain nor of any operation except tonsillectomy at the age of 7 years. She had never passed dark urine. Her mother was alive but the father had died 2 years previously from a cerebral haemorrhage. She was not aware of skin fragility in either of her parents.

Specimens of urine and faeces were requested from both parents and a younger brother of the patient. All three urines were negative on spectroscopic examination for porphyrin. The faeces of father and brother contained coproporphyrin 7 and 6, protoporphyrin 37 and 20μg. per g. dry weight respectively, i.e. within normal limits, but that of the mother contained copro- 51, proto- 149, which are definitely increased.

Liver function tests were repeated on the child on 8 March and 15 May 1956, with results similar to those reported above.

DISCUSSION

This case is of unusual interest for two reasons, (*a*) the close superficial resemblance to a case of congenital porphyria and (*b*) the unusually early appearance of cutaneous manifestations in comparison with other South African cases of familial porphyria.

Gunther (1911, 1922) made a distinction between the cases of cutaneous porphyria in which the lesions commenced in babyhood, eventually resulting in extensive scarring and mutilation (porphyria congenita), and those in which similar but milder lesions commenced later in life (porphyria chronica). Since some patients in the latter group complained at times of gastro-intestinal disturbances he suggested that this form of porphyria might be intermediate between the congenital and the acute forms. Waldenström (1937) separated the congenital type from the chronic and renamed the latter porphyria cutanea tarda in order to emphasize their salient difference, but maintained that the occasional occurrence of colic in cases of cutanea tarda indicated a possible relationship with the acute type of porphyria. This subdivision of cutaneous porphyria according to age of onset of lesions was ignored by Turner and Obermayer (1938), who collected 86 cases they considered to be congenital. The ages of onset, where recorded, in their series were: antenatal in 7, 0-10 years in 36, 10-20 years in 6, and above 20 in 19. Familial incidence was recorded in 30 cases.

Watson *et al* (1951) then showed that cases of porphyria could be divided into 2 main groups, the erythropoietic, when the site of the chemical disturbance was in the bone marrow, and the hepatic, when it was in the liver. Those of the cases formerly called congenital which conformed most closely to the classical description belong to the erythropoietic group, and the other cases, subdivided into intermittent acute, cutanea tarda, mixed or intermediate, and latent forms, fall into the hepatic group. The cases listed by Turner and Obermayer, and also the cutaneous cases subsequently published, were critically reviewed by Schmid, Schwartz and Sundberg (1955). Only 36 of these satisfied the criteria they laid down for erythropoietic porphyria; other cases may have belonged but had to be excluded because of inadequate information.

The case we have described is excluded from the erythropoietic group because of normal findings in bone-marrow films. The patient is far from robust and a liver biopsy was not considered to be justified. The stool porphyrins differ from those found in congenital porphyria but agree with the results obtained in many other cases of familial porphyria in South Africa.

In these studies there is no record of clinical manifestations in White children. Dean and Barnes (1955) did not find detectable excess of porphyrin in the urines of any members under 20 years in the large family they reported. It has, however, been found recently (Barnes, unpublished data) that some of the children with one affected parent excrete more than normal amounts of porphyrin in their faeces but none of these gave values as high as were found in this child.

Some of the younger patients in Turner and Obermayer's list not regarded as erythropoietic porphyria were probably cases of the hepatic form. MacGregor, Nicholas and Rimington (1952) reported a case of porphyria cutanea tarda in an English boy whose lesions had commenced at the age of 11 years. Schmid, Schwartz and Watson (1954) state that one of their patients in the hepatic group 'had rather atypical clinical features, especially in an onset of photosensitivity at the age of 5 years, much earlier than is usual in this group and even belying the designation cutanea tarda'.

Our patient, considered solely on the findings in his own family group, would be regarded as an example of the cutanea tarda form of hepatic porphyria, though the age of onset belies the designation even more than it does in Schmid's case. On the other hand he may be a case of hepatic porphyria of the familial type common in South Africa, which should be regarded as the mixed form in which cutaneous and acute symptoms both occur.

We have to thank the Director of the Institute for his interest in this work and for providing the necessary facilities. Dr. H. B. W. Greig carried out the haematological examinations, including the fluorescence study of the bone marrow. The photographs were taken by Mr. Ulrich of the Photographic Department of the South African Institute for Medical Research.

A description of this case was included in the thesis submitted by H.D.B. to the University of London for the Degree of Doctor of Philosophy.

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POLIOMYELITIS VACCINE MAY BE IMPORTED FROM ISRAEL

BY OUR PARLIAMENTARY CORRESPONDENT

The Israeli Government may be in a position to export poliomyelitis vaccine and the possibility of obtaining supplies from that country is being explored through the good offices of the Envoy Extraordinary and Minister Plenipotentiary for Israel in the Union, according to an announcement made by the Minister of Health (Mr. J. H. Viljoen) in the House of Assembly on 26 March 1957.

The Minister also announced that he had been informed that supplies of vaccine from America were unlikely to become available for export before the middle of April. Confirmation had been received from the Union's Embassy in Washington that this position obtained in relation to all vaccine manufacturing firms in the United States.

Mr. Viljoen, who was replying to a question, said his attention had been drawn to a press report of 16 March in which it was denied that the Union Government had applied to the American Department of Commerce—stated in the report to be the licensing authority in the United States—for export permits with a view to the importation of poliomyelitis vaccine into the Union.

After reiterating the steps taken by the Union Government to obtain supplies through normal trade channels when it transpired that the American Government was not prepared to supply vaccine on an intergovernmental basis, Mr. Viljoen said the press report referred to apparently arose from the transmission of inaccurate information to America.

'It is unfortunate that in spite of assurances regarding the importation of vaccine given by me in a reply to questions put from time to time, this matter was ventilated in this House with so much misunderstanding, culminating in a press report which could have prejudiced the issue of licences for the export of vaccine to the Union', he said. 'I viewed this possibility in such a serious light that as soon as the report was brought to my notice, I immediately instructed my Department to request the Union's Embassy in Washington by cablegram to correct any possible misunderstanding. I also made the position clear to the United States Ambassador in the course of a personal interview on 18 March on which occasion his attention was invited to the statement made by me in this House during the relative debate, when I indicated that it was not for me to instruct the United States Government how it should act in connection with the export of vaccine.

'According to the latest information received the necessary export licence was granted to the manufacturing firm in America, with whom the Union's order for vaccine had been placed, and has been in the firm's possession since the week before last. From this it is abundantly clear that the relative report was not based on fact.'

Replying to a question by Mr. L. Whiteley (Durban-Umbilo), the Minister said that according to preliminary statistics, the mortality rate per 1,000 births during 1956 was Europeans 31, Coloureds 136·5 and Asiatics 66·3. As the registration of Native births was still very incomplete, no reliable statistics were at present available on the infant mortality rate for Natives.

THE AFRICAN DIET AND THE HEALTH OF THE CHILDREN

DR. F. J. C. CAMBOURNAC

Director of WHO for Africa World Health Day

The health of the children is a key asset for Africa. Mothers know this well and take care of it to the best of their ability. However, what African mothers do not know is that the health of their children is jeopardized not only by diseases, but also by another alarming threat:

MALNOURISHED DOES NOT MEAN UNDER-FED

Malnutrition and under-feeding are not to be confused. One cannot say that Africa suffers from hunger, it is rather a malnourished continent. As a corollary to an ill-balanced diet, and lack of sufficient knowledge, certain basic elements indispensable to the body stamina and the maintenance of health are generally missing in the food, or if present, are available in too small quantities.

Among these essentials, those which are most frequently absent in African diet are proteins, especially animal proteins, and vitamins. A deficiency of these nutritional elements may cause severe symptoms, such as *kwashiorkor*, which may lead to death; it may also considerably aggravate morbidity caused, for instance, by malaria, bilharzia or any other tropical parasitism; finally, it may so debilitate the human body as to make it an easy prey for disease.

THE MOST VULNERABLE GROUP

Pregnant women and children are naturally the most vulnerable group to these deficiency diseases. In the least developed areas, this group represents up to the 40% of the population. Childhood and pregnancy are the periods during which the human organism is most exposed to disorders caused by dietary deficiency. Therefore, it is during childhood, pregnancy and breast-feeding that the diet must be adequate, so as to ensure a better resistance to these conditions. A well-balanced diet will guarantee to the mother a healthy baby. Similarly, her child will grow up to be a vigorous adult. Therefore, nutrition is one of the most important public health problems in Africa.

HOW TO TACKLE THE PROBLEM?

This problem is to be tackled from three different angles:

1. In order to have an improved knowledge of the situation within the continent, and of the incidence of malnutrition, it is necessary to carry out epidemiological studies which should reveal the cause and frequency of nutritional diseases.

2. A study of local foods and food preparation is indispensable, in order to ascertain their actual food value.

3. It is obvious that in most parts of Africa, beliefs and superstitions are opposed to the intake of some beneficial items . . . such as milk, for instance. Suitable health education programmes must therefore be established, and brought within the understanding of the mothers, so as to enable them to better realize the relationship which exists between food and the health of their children. Public health auxiliary personnel, on the other hand, should also be specially trained. The best method for the latter to approach the subject is to teach the mothers while treating their children, so as to improve their understanding, through example, of the relationship between the cause and the effect of malnutrition.

NUTRITIONAL REHABILITATION CENTRES

It seems that one of the best methods of controlling malnutrition would be the setting up of 'Nutritional Rehabilitation Centres' where mother and child Health Centres already exist. Malnourished children would be committed to the care of these centres, in serious cases, as in-patients. In minor cases they would stay during the daytime and thus would give the staff the opportunity of keeping in constant touch with the mothers, who would be taught the diets for the children and the methods of

preparation. The care of the children would thus be augmented by the education of the parents.

In some places, the lack of funds might make it impossible to set up such centres, but it would be desirable to create at least a small pilot-project, the findings of which might be applicable to a much larger area.

Another task of the centres would be to supervise the diet of pregnant women, for everybody knows how much the health of the new-born depends upon the condition of the mother-to-be.

A GOOD START IN LIFE

The increasing of good quality proteins in the diet of the pregnant and of the breast-feeding mother is the best prevention against the ill effects of malnutrition in children. Not only will the child be less susceptible to disease, but it will be fed a longer time by its mother, and thus will receive longer protection. It will then be in a stronger condition to meet the most delicate period of its feeding, that which immediately follows weaning, often characterized by *kwashiorkor*, the dangerous symptoms of which are a consequence of the change from the breast to adult food lacking in proteins.

The activity of WHO in the field of nutrition is threefold and consists of: (1) the training of personnel, (2) the fostering of research, (3) the assistance to governments.

Two nutrition courses organized jointly by the French government, FAO and WHO, in 1952 and 1955 in Marseilles were attended by numerous participants who are today practising in the field. It is expected that a similar course will take place in English some time in 1957. Within the last 3 years, a total of about 20 fellowships have been granted to enable their holders to improve their knowledge in the field of nutrition.

WHO works in close relationship with governmental institutions entrusted with the study of nutrition problems in Africa, and particularly with the discovery of new sources of proteins. Finally, at the request of the governments concerned, WHO has sent consultants and specialists into the following countries: Basutoland, Northern Rhodesia, Southern Rhodesia, Mauritius, Uganda and Kenya. The aim of their work is to assess the present situation and to propose means to improve, thanks to better nutrition, the health of the Africans.

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PASSING EVENTS : IN DIE VERBYGAAN

Admission to Closed Hospitals. Members are reminded that the position in regard to the production of certificates of inability to pay for private medical attention remains unchanged at the 'closed' (Teaching) Hospitals.

* * *

Nuffield Dominion Travelling Fellowships for 1958. Applications are invited *not later than* 30 April 1957, for the following senior fellowships offered to South African nationals by the Nuffield Foundation for the year 1958: One fellowship in the Natural Sciences. Two fellowships in Medicine. One fellowship in the Humanities. An additional fellowship may be awarded in any one of these fields.

Applicants should normally be between 25 and 35 years of age, have high intellectual and personal qualities, and have shown capacity of an unusual order for advancing knowledge and education in their fields. They must undertake to return to South Africa as soon as possible after the termination of their fellowships.

Copies of the regulations and application forms may be obtained—(*a*) *by University applicants:* from Registrars of their

Universities. (*b*) *by others:* from the Honorary Secretary, Nuffield Foundation South African Advisory Committee, c/o University of the Witwatersrand, Johannesburg.

Applications received after 30 April 1957, will not be considered until 1958.

* * *

Dr. Harold C. Hofmeyr of Cape Town will be leaving on 2 April for a 4-month visit to Europe. He will visit hospitals and clinics in England, Germany and Sweden. He will also attend the Annual Conference of the BMA and meetings of the American College of Chest Physicians.

* * *

Mr. Hugh Benjamin, F.R.C.S. (Edin.) has returned from America after visiting clinics in Boston, Chicago and New York.

* * *

Mr. M. Modlin, F.R.C.S. (Eng.) has commenced practice as a urologist at 406/7 Medical Centre, Cape Town. He has been in the United Kingdom for almost 5 years, during which time he worked in the Department of Surgery, Hammersmith Hospital, Post-Graduate Medical School of London, and as Surgical Registrar in the Department of Urology, University of Leeds, the General Infirmary and St. James' Hospital, Leeds. Telephones: Rooms 30154, Residence 47034.

Dr. H. P. J. Pretorius, M.Med. (Paed.), formerly of the Department of Paediatrics, General Hospital, Pretoria, has joined Dr. J. J. Theron, Paediatrician, in practice at 215 Lister Building, Jeppe Street, Johannesburg. Telephones: Rooms 22-0614, residence 41-4769. Dr. Pretorius has recently returned from New York City where he worked in the Pediatric Department at the Bellevue Medical Center under Dr. L. Emmett Holt, Jnr.

Dr. H. P. J. Pretorius, M.Med. (Paed.), voorheen van die Departement van Kindergeneeskunde, Algemene Hospitaal, Pretoria, praktiseer nou saam met dr. J. J. Theron, Kinderarts, te Listergebou 215, Jeppestraat, Johannesburg. Telefone: Spreekkamers 22-0614, woning 41-4769. Dr. Pretorius het onlangs vanaf New York teruggekeer waar hy onder dr. L. Emmett Holt, Jnr. by die Bellevue Medical Center gewerk het.

* * *

Members are reminded that they should notify any change of address to the Secretary of the Medical Association of South Africa at P.O. Box 643, Cape Town, as well as to the Registrar of the South African Medical and Dental Council, P.O. Box 205, Pretoria. Failure to advise the Association can only result in non-delivery of the *Journal*. This applies to members proceeding overseas as well as to those who change their addresses within the Union.

REVIEWS OF BOOKS : BOEKRESENSIES

PHYSICAL MEDICINE

Annals of Physical Medicine. Volume IV—Number 1—February 1957. Editor: A. C. Boyle. Pp. vi + 40. Annual Subscription £1 10s. Quarterly 10s. London: Baillière, Tindall and Cox Ltd. 1957.

Contents: Editorial. The Presidential Badge. *Revision Series*. The Theory and Practice of Electrodiagnosis, by A. T. Richardson and C. B. Wynn Parry. *Original Paper*. Physical Medicine and Modern Physics, by Svend Clemmesen. *New Appliances*. An Apparatus for Buerger's Exercises in the Home, by J. B. Jessiman. The International Congress of Physical Medicine, Copenhagen, 1956. British Association of Physical Medicine. North and Midlands Physical Medicine Club. Harveian Tercentenary Congress, 1957. Reviews of Books. Abstracts of the Literature.

This publication which has just been taken over by Messrs. Baillière, Tindall & Cox, Ltd., is the official journal of the British Association of Physical Medicine. It continues the splendid contributions which have been made in the former numbers. In its new format, re-styled and enlarged, it should be of great interest to those who are practising and are interested in the subject. Another innovation is the publication of the first article in a Revision Series, which the editor hopes will become a regular feature of the Journal.

T.S.

THERAPEUTICS

A Therapeutic Index. Second Edition. By C. M. Miller, M.D. (Lond.), M.R.C.P. (Lond.) and B. K. Ellenbogen, M.D. (L'pool), M.R.C.P. (Lond.) with a Foreword by E. Noble Chamberlain, M.D., M.Sc., F.R.C.P. Pp. xii + 156. 12s. 6d. London: Baillière, Tindall and Cox Ltd. 1957.

Contents: Therapeutic Index. I. Cortisone and Corticotrophin. II. Dosage Tables. III. Practical Procedures. IV. Simple Urine Examinations. V. Some Useful Diets. VI. Infectious Fevers. VII. Normal Values. VIII. Approximate Equivalents and Conversion Factors.

The popularity of this book has made this 2nd edition necessary and the recent advances in medicine have added to the necessity for bringing its contents up to date.

Its pages summarize the main points of modern treatment and it is amazing how the authors have managed to get so much into such a small compass.

In addition to the index of treatment, it contains much valuable information in the way of practical procedures, diet tables and periods of incubation, isolation and quarantine. There are also chemico-therapeutic and antibiotic-dosage charts.

This book should be of particular value to young practitioners and is likely to prove its value frequently, particularly in cases of emergency.

A.T.

PSYCHOLOGY AND RELIGION

Psychology, Religion, and Human Need. A Guide for Ministers, Doctors, Teachers, and Social Workers. By W. L. Carrington, M.D. Pp. xi + 315. 30s. net. London: The Epworth Press. 1957.

Contents: Introduction. *Section I. The Modern Psychological and Religious Approach to Human Need*. 1. The General Nature and Scope of Personal Help. i. The Modern Christian Ministry. ii. The Inner Resources Needed. iii. Making Contact and Winning Confidence. 2. The General Psychological Background. i. The Springs of Human Attitude and Conduct. ii. The Main Schools of Psychology. 3. The Specific Psychological and Religious Background. Development of Personality, Abundant Life. *Section II. The Personal and Educational Ministry*. 4. The Fine Art of Visiting and Interviewing People. i. Is Pastoral Visiting still worth while in these days? ii. The Real Purpose of Pastoral Visiting. iii. Some Practical Aspects of Visiting. iv. The Fine Art of Interviewing. v. Some Practical Aspects of Interviewing. 5. Helping People in Worship, Prayer, and the Guidance of God. 6. Educating People for Christian Marriage and Parenthood. i. An Urgent and Universal Need ii. A Comprehensive Plan to Meet it. 7. Pre-Marital Preparation: Medical and Pastoral Aspects. i. The Medical Part in Pre-Marital Preparation. ii. The Pastoral Part in Pre-Marital Preparation. 8. The Wedding and the Early Years of Marriage. i. Making the Most of the Marriage Service. ii. Education After Marriage and For Parenthood. 9. Equipping Parents to give Religious and Sex Education to Young Children in the Home. i. Example: The Character of the Parents. ii. Experience: The Parents' Control of the Child's Environment. iii. Experiment: Project and Practice. iv. Direct Teaching by Picture, Word, and the Answering of Questions. v. The Sexual Part of Religious Education in the Home. 10. Equipping Parents and Others to Understand and Help the Adolescent. Fitness for Marriage, Courtship and Choice of Mate, 'The Case for Chastity'. *Section III. The Healing Ministry*. 11. The Church's Part in Healing. 12. Priestly Healing. 13. Pastoral Healing through Personal Counselling. 14. The Christian Approach to some Emotional Disorders. i. Resentment against Life, or against God. ii. Resentment against Other People. iii. The Sense of Guilt of Failure. iv. Anxiety and Apprehensiveness. v. Frustration and Futility. vi. Loneliness. vii. Grief. viii. Some Sexual Difficulties and Conflicts. 15. Understanding and Helping the Alcoholic. 16. Understanding and Helping the Mentally Ill. i. What do we mean by 'Mental Illness'? ii. The Psychoneurotic and Psychosomatic Reaction Types. iii. The Psychotic Reaction Types. iv. Psychopathic Disorders. 17. Marriage Counselling. 18. Visiting the Sick. *Section IV. The Evangelistic Ministry*. 19. 'Every Christian an Evangelist'. Index.

Under this impressive title Dr. W. L. Carrington writes a competent book for Protestant Evangelicals who have keen interest in church work.

Ten per cent of the volume is a useful summary of Freud, Yung, Adler, Flugel, Watson, Horney, du Nouy; all of them subordinated

to the writer's personal concern touching pastoral work. The remainder of the book, 18 chapters on ministerial work, is thorough, if too comprehensive. The chapters on Some Emotional Disorders and The Mentally Ill will guide the young pastor, but the relationship of modern medicine and orthodox Christian theology needs more thought than this book suggests. Wisely the writer takes the normal medical point of view, touching Priestly Healing, but if the psycho-somatic is about to intrude on hospital practice, the when, how, and to what degree, will demand thought far beyond evangelical conviction: worthy as this latter is.

M.R.

PHYSIOLOGY AND PATHOLOGY IN KIDNEY DISEASE

British Medical Bulletin. Vol. 13, No. 1 (January 1957). Scientific Editor, Clifford Wilson, M.A., M.D., F.R.C.P. Pp. 74. £1 per number, £2 10s. 0d. per volume. London: Medical Department, The British Council.

Contents: Seventeen articles on the Physiology and Pathology of the Kidney by the following authors: D. A. K. Black and E. W. Emery, E. M. Darmady and Fay Stranack, M. A. Floyer, H. Harris, G. C. Kennedy, J. M. Ledingham, R. A. McCance and E. M. Widdowson, Mary G. McGeown and Graham M. Bull, C. R. Macpherson and A. G. E. Pearse, M. D. Milne, R. C. Muehrcke and B. E. Heard, R. S. Murray and G. C. Tresidder, John R. Squire, J. D. Blainey and J. Hardwicke, S. W. Stanbury, R. E. Steiner, Clifford Wilson and D. Gordon Abrahams, Oliver Wrong.

This monograph represents another of the British Council's Symposia on the advance of medical research in Great Britain. It is a most valuable reference monograph for those who are interested in the application of Physiology and Pathology to the clinical problems of kidney disease and disturbances of the body's homeostasis in metabolic disorders.

In the introduction Professor Robert Platt explains why the monograph is written by clinicians rather than by physiologists and pathologists. He claims, we think rightly, that the exciting progress of medicine over the last 25-30 years largely arises from the application of physiology to clinical medicine. He states that three outstanding methodological discoveries have influenced recent work on the kidney; namely flame photometry, chromatography and the discovery of aldosterone.

It would be difficult to single out any one of the 17 articles for special mention but undoubtedly the last, on the effects of over-nutrition on the kidney, is extremely stimulating to new thought.

J.F.B.

CORRESPONDENCE : BRIEWERUBRIEK

‚MENEER' OR 'DR'

To the Editor: The letter by Dr. J. H. van Eck Kirsten[1] is typical of those who forget the great history and traditions of Medicine by concern over, ‚'n egte Suid-Afrikaanse lewenswyse'. It so happens that the 'Mother' Medical Schools of South Africa, namely, Cape Town and Witwatersrand, were initially mainly staffed by professors and lecturers from Great Britain, and the fine traditions which they have planted here (and which we may now conveniently call 'South African') have been the solid foundations on which a medical ‚egte Suid-Afrikaanse lewenswyse' has developed and blossomed. It is the 'Misters' of South African medical schools that have trained Dr. Kirsten, and to wish to unshackle oneself from such traditions now, is to forget South African medical history.

The practice and art of medicine is well above racial and national barriers. Medicine is a truly world-wide fraternity with its own code of ethics and traditions, and Dr. Kirsten may well decide to re-orientate his concepts.

Finally, it would seem wholly discourteous to write from the Royal College of Surgeons Club in a manner derogatory to one's hosts.

Ronald Singer

Medical School
Mowbray
Cape Town
25 March 1957

1. Kirsten, J. H. van Eck (1957): S. Afr. Med. J., **31**, 296.

ANEURISMA VAN ABDOMINALE AORTA

Aan die Redakteur: Ek wens te verwys na die artikel deur dr. J. K. Bremer oor Aneurisma van die Aorta in die uitgawe van u blad gedateer 9 Maart.[1] Nie alleen is dit 'n relaas van die uitmuntende werk van dr. Bremer nie, maar dit bepaal die aandag van die geneeskundige beroep in Suid-Afrika op die groot vooruitgang wat daar gemaak is met die snykundige behandeling van hierdie ernstige arteriële siekte. Veral belangrik is die feit dat selfs 'n lekkende aneurisma van die aorta vandag met sukses opereer kan en moet word.

Alhoewel my persoonlike ondervinding van die vervanging van 'n aneurisma van die abdominale aorta met 'n kunsmatig-voorbereide prostese tot slegs 2 gevalle beperk is, wil ek tog 'n toevoeging tot die operatiewe tegniek, wat ek in een pasiënt probeer het, aanbeveel. Dit is 'n metode om die aanhegting tussen die prostese en die aorta te versterk by 'n pasiënt waar die aorta by die reseksie-punt só ernstig beskadig is, dat daar vrees moet ontstaan of die hegtings nie sal uitskeur nie.

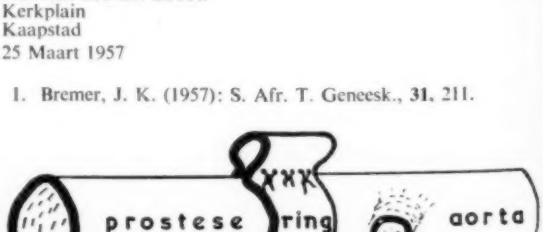
By my pasiënt het die bloedstolsel sover proksimaal versprei tussen die intima en die media van die aorta—en dit kon selfs pre-operatief op die X-straal-foto's bepaal word—dat daar nie genoegsame redelik gesonde aortawand onderkant die renale vate oorgebly het om 'n stewige hegting aan te bring nie. Nadat groot stukke van die intima verwyder is, is die hegtings-poging belemmer deur die feit dat die hegtingsnaald eenvoudig deur die res van die aortawand geskeur het. Om te voorkom dat die aanhegting tussen die aorta en die poliviniel prostese in flenters skeur, het ek 'n poliviniel buis gemaak met wyer deursnit as die poliviniel prostese en dit as 'n versterkende omhulsel oor die prostese, wat toe nog nie distaal geheg was nie, geskuif. Oombliklik na die verwydering van die proksimale aorta-klem was ek in staat om hierdie omhulsel op te skuif tot teenaan die renale vate, dit styf om die lasplek te trek en dit met behulp van 'n arterieklem, nog stywer vas te trek om die aanhegting tussen prostese en aorta. Met die klem nog in posisie, was ek in staat om 'n paar hegtings deur die plooi van hierdie omhulsel te steek, en aldus te verseker dat dit die anastomose stewig omring nadat die arterieklem verwyder is. Op hierdie wyse kon ek nie slegs die onvermydelike onmiddellike bloeding by die naat beheer nie, maar ook 'n latere deurbarsting voorkom.

Aangesien 'n mens altyd gereed moet wees om 'n prostese in die operasiesaal te vervaardig, is die materiaal tog byderhand om so 'n ekstra omhulsel, wat wyer as die prostese moet wees, te maak. Al sou hierdie prosedure geen ander waarde hê nie, gee dit mens tog 'n gevoel van veiligheid.

F. D. du T. van Zijl

National Mutual-Gebou
Kerkplain
Kaapstad
25 Maart 1957

1. Bremer, J. K. (1957): S. Afr. T. Geneesk., **31**, 211.



Die tekening toon die ring of omhulsel nadat dit styf om die naat getrek was en met drie hegtinge vasgetrek is.